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CLINICAL LECTURES

DISEASES OF THE HEART AND AORTA

G. W. BALFOUR

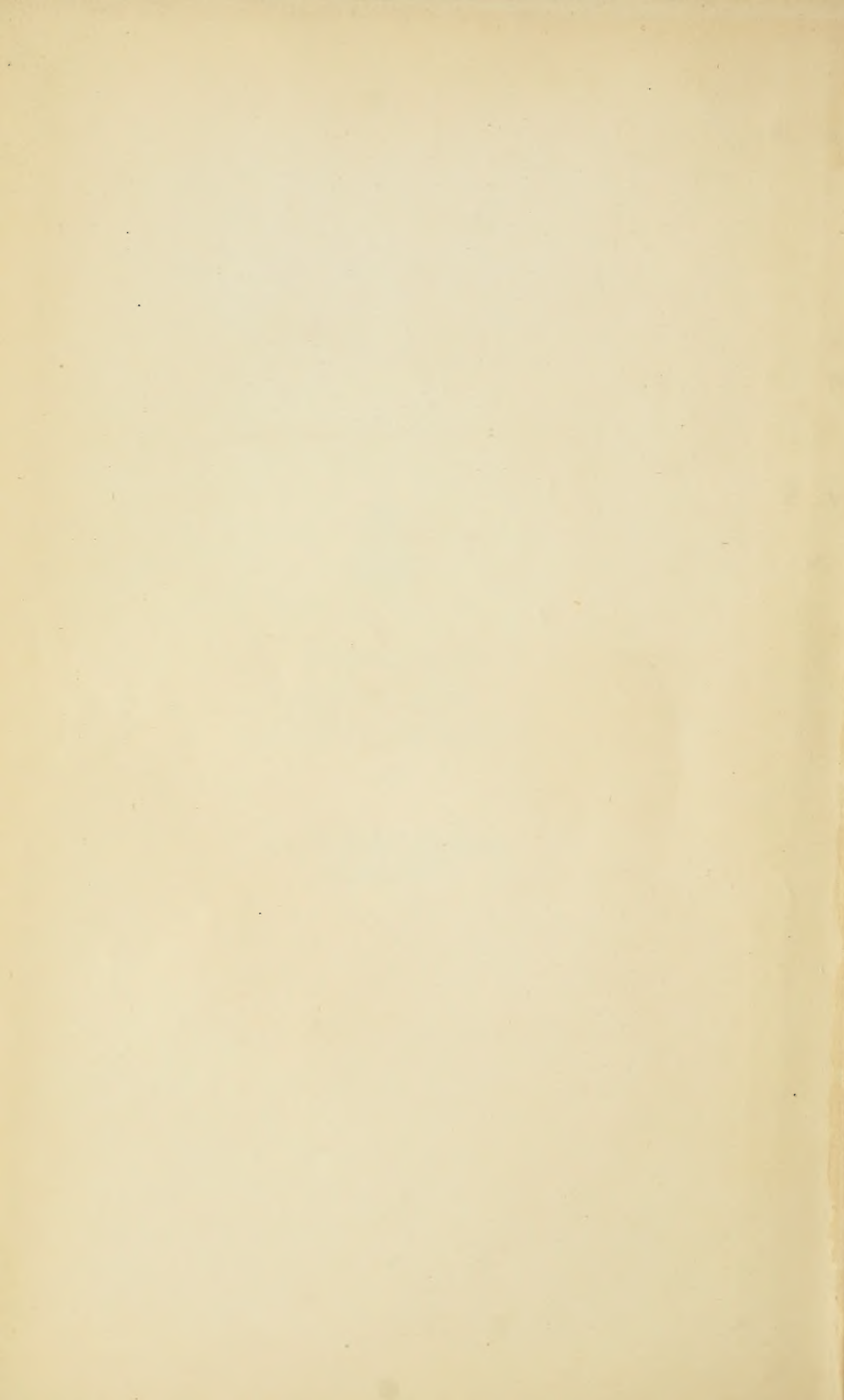


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AND AORTA

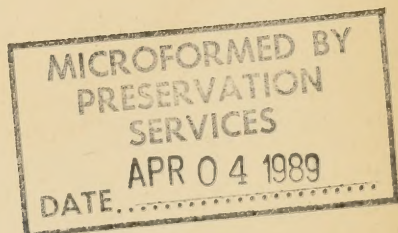
BY

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THIRD EDITION



LONDON  
ADAM AND CHARLES BLACK  
1898

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CLINICAL LECTURES

ON

DISEASES OF THE HEART AND AORTA







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TO THE

**Most Honourable the Marquess of Bute, K.T.**

LL.D. of the Universities of St. Andrews  
Edinburgh, and Glasgow

RECTOR OF THE UNIVERSITY OF ST. ANDREWS  
Etc. Etc. Etc.

AS A TOKEN OF THE RESPECT AND ESTEEM

ENTERTAINED FOR HIM AS A MAN OF LEARNING

A PATRON OF LEARNING

AND A WARM FRIEND TO THE UNIVERSITY OF ST. ANDREWS

BY THE

AUTHOR



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1898



## PREFACE TO THE THIRD EDITION

ADVANTAGE has been taken of the present opportunity to revise and entirely rewrite these Lectures. A considerable amount of new matter has been incorporated, and it is hoped that this new edition may be found worthy of the favour with which former editions were received.

EDINBURGH, *December* 1897.

## PREFACE TO THE SECOND EDITION

I HAVE to thank the profession for the kindly manner in which the first edition of this work has been received, notwithstanding its many faults. The present edition has been almost entirely rewritten and is somewhat enlarged, advantage having been taken of this opportunity to obviate certain misconceptions to which some of the statements in the former edition seem to have been exposed. The living cases formerly narrated have been brought down to date, one or two having fallen out of sight. An attempt has also been made to explain the action and to formulate rules for the use of *digitalis* in disease of the heart, and of iodide of potassium in aneurysm, which, if not absolutely accurate, are at least in accordance with the results obtained by pharmacologists, and will, it is hoped, prove useful to practitioners. As formerly, I desire all my statements of facts, or what I believe to be facts, to be carefully tested; but as no human work is perfect, I claim indulgence for the manner of stating.

EDINBURGH, *December* 1881.

## PREFACE TO THE FIRST EDITION

“I hold every man a debtor to his profession ; from the which, as men of course do seek to receive countenance and profit, so ought they of duty to endeavour themselves, by way of amends, to be a help and ornament thereunto.”—*The Works of Francis Bacon* (London, 1859), vol. vii. p. 319.

WITH Latham I may say that mine is but “a limited purpose—it is to regard the diseases of the heart only in one point of view, *i.e.* as they appear in the living man.” As this is the object of clinical teaching, I have called these lectures clinical, though otherwise the term is somewhat of a misnomer, as the illustrations have been mainly taken from patients who have passed from the clinique into the domain of morbid anatomy. One obvious advantage of this method is the definite connection, provided the cases are sufficiently numerous, of certain distinct *pre-mortem* phenomena with equally well-defined *post-mortem* appearances; a sufficient guarantee for the accuracy of this connection being to be found in the publicity inseparable from a large teaching hospital like the Edinburgh Infirmary, and also in the fact that the life history is closed by the physician in the case-book before the pathologist commences his record of the morbid appearances. In a work such as the present it was impossible to avoid narrating cases, but I have restricted these to as few as possible, giving only the histories of those patients who may be regarded as affording well-marked examples of the phenomena attending any given lesion.

No two cases of any disease are ever exactly alike, even though depending on a similar lesion. The object of all clinical teaching is to show how we are led to determine what is the central lesion in any given case, and to explain how the apparently inexplicable phenomena, which constitute what we term the disease, group themselves naturally round that lesion in accordance with physical and physiological laws; and, finally, to show how we can influence that central lesion by remedies, and how effectually this modification of the cause modifies also the concomitant phenomena, though now and then some of these become so developed as to require special and independent treatment, a treatment which can never be anything but palliative, while that of the central lesion may not infrequently be really curative. All this I have endeavoured to do in relation to cardiac disease, and though none of the lectures have ever been actually delivered as they now stand, they yet comprise the essence of my clinical teaching in regard to diseases of the heart and aorta during the last eight years. By putting this into lecture form, I have been enabled to write with more ease to myself, and, what is of more importance, I have been enabled to avoid noticing any special subject which has not happened to come under my own observation. I have thus been enabled to avoid the mere repetition of the statements of those who have preceded me in the same field of inquiry, and by transcribing my descriptions of disease directly from the book of nature, and basing my explanations of the phenomena observed—and, to a large and most important extent, their treatment also—upon physical laws and the results of experimental physiology,<sup>1</sup>

<sup>1</sup> I may as well state here, that though I myself never performed one single experiment upon a living animal, and though I deprecate as much as any one can the undue multiplication of such experiments, I yet hold it to



I have done what I could to remove the diagnosis and treatment of these diseases from the domain of mere speculative opinion, and to place them under direct scientific control. And if in any respect I have failed in doing so, the failure is undoubtedly due to my own ignorance, and will by and by be remedied by some worthier successor, who will base his diagnosis upon a more successful application of the laws and facts of physics to the explanation of the phenomena connected with cardiac disease, and rest his treatment upon more accurate physiological knowledge; for "the thoughts of men are widened by the process of the suns." To trace this process in the diagnosis and treatment of cardiac disease we neither require to go very very far back in medical history nor even to read many books. For though in 1531 the pulmonary circulation was shown by Servetus to be reasonably probable,<sup>1</sup> yet it was not till 1628—nearly a century afterwards—that Harvey first gave to the world a full account of the double systemic and pulmonary circulation, which he had already publicly taught and demonstrated since 1619.<sup>2</sup> Of course any attempts at the diagnosis of cardiac diseases previous to this period can possess no scientific value whatever, and even their treatment must have been founded on the vaguest empiricism. For long after this era, as before it, the various essays upon diseases of the heart are of no value except

be true that much that is now plain in cardiac diagnosis, and definite in the treatment of cardiac disease, has been made so by means of experimental physiology, and that many patients now living owe their lives primarily to this method of investigation, a method of investigation which, kept within due bounds, cannot be regarded as inconsistent with Christian humanity, when we reflect that to save one life alone a whole herd of swine were sent into the depths of the sea of Gennesareth, and that we are also told "ye are of more value than many sparrows," or frogs either.

<sup>1</sup> *De Trinitatis Erroribus*, Basil, 1531.

<sup>2</sup> *Exercitatio Anatomica de Motu Cordis et Sanguinis in Animalibus*, Francoferæ, 1628.

in so far as they contain a number of curious pathological observations. Even Senac's great work, the first complete monograph upon diseases of the heart, is solely valuable in this respect.<sup>1</sup> Thus, while Harvey may be considered to have founded the modern physiology of the heart and the circulation in 1628, and Morgagni must be equally regarded as the founder of the modern pathology of the organs concerned in 1762,<sup>2</sup> it is only from Corvisart that we date the origin of the modern system of diagnosis of their diseases in 1806.<sup>3</sup> Corvisart was first physician to the great Napoleon, and was said to have been the only man of independent mind about his court, having been not more remarkable for his modesty than for his skill, especially in diagnosis. As a sample of the high estimation in which his diagnostic powers were held both by the laity and the profession, two anecdotes may be related. In one he is represented as standing before a portrait and saying, "If the artist painted this picture correctly from the life, the original of this portrait must be already dead from disease of the heart." It is added that this was actually the case. In the other, Dupuytren is represented as saying, "I have often observed the marvellous skill with which Corvisart not only pointed out the nature and position of a cardiac lesion, but also indicated almost to a line the exact size of that cardiac orifice he had diagnosed as contracted." We know very well that no amount of diagnostic skill would, even nowadays, justify either statement, though by a species of *εὐστοχία* Corvisart might by chance have been right enough in occasional instances.

<sup>1</sup> *Traité de la Structure du Cœur, de son Action, et de ses Maladies*, Paris, 1749.

<sup>2</sup> *De Sedibus et Causis Morborum per Anatomea indagatis*, Venet. 1762.

<sup>3</sup> *Essai sur les Maladies et les Lésions Organiques du Cœur et des gros Vaisseaux*, Paris, 1806.

He was the first to make practical use of Auenbrugger's invention (1761) of percussion,<sup>1</sup> and he was also in the habit of listening to the sounds made by the heart; but it was to Lænnec, as every one knows, that we owe the first (1819) attempt at the scientific application of auscultation to the diagnosis of disease.<sup>2</sup> And it is to Bouillaud (1824) that we owe the first application of scientific auscultation to the diagnosis of cardiac disease,<sup>3</sup> thus opening up the means of attaining an accuracy, the possibilities of which are as yet only dawning on the professional mind. But it is to the clinical experience, and, above all, to the practical experiments of James Hope (1832),<sup>4</sup> that we owe a very considerable advance in our knowledge of the causes of cardiac murmurs in particular. I believe he is the first who noted the occurrence of mitral regurgitation from simple dilatation of the ventricle, the orifice being healthy. To Bouillaud, however, following Lænnec, we owe the distinct statement that the auricle is found to pulsate alternately with the ventricle in certain cases of disease, particularly in cases of mitral constriction. And this observation, which is altogether slighted by Hope, had no doubt its influence in leading M. Forget (1851)<sup>5</sup> to his doctrine of retro-dilatation,<sup>6</sup> which is the first distinct, though very imperfect attempt at the foundation of a physiologico-pathology of the morbid heart, a mode of investigation which seems destined to produce very important results in the detection of cardiac diseases. Closely

<sup>1</sup> *Inventum Novum ex Percussione Thoracis humani, ut signo abstrusos pectoris morbos detegendi*, Vindobonæ, 1761.

<sup>2</sup> *De l'Auscultation médiate*, Paris, 1819.

<sup>3</sup> *Traité Clinique des Maladies du Cœur*, Paris, 1824.

<sup>4</sup> *Treatise on Diseases of the Heart and Great Vessels*, London, 1832.

<sup>5</sup> *Précis Théorique et Pratique des Maladies du Cœur*, Strassbourg et Paris, 1851.

<sup>6</sup> *Op. cit.* p. 18.

following Forget, we have the very instructive work of Stokes<sup>1</sup> (1854), whose interesting chapters upon the condition of the heart in fevers, and upon the effect of defective cardiac power upon the cerebral circulation, are amongst the most important additions made in recent times to our knowledge of cardiac pathology. The only other work that I have to add to this list, fully to bring down the history of cardiac diagnosis to the present day, is the admirable compendium of Von Dusch<sup>2</sup> (1868), which has, in my opinion, no equal in any language as a practical exposition of the art of diagnosing diseases of the heart.

It is not yet seventy years since science in any form became applied to the diagnosis of cardiac disease; each of the works which I have enumerated constitutes an era in the history of the development of this diagnosis, and a consideration of the half-dozen works I have recorded is sufficient to enable us to trace the progress of cardiac diagnosis from its earliest dawnings quite down to the present day. To mark more accurately the gradual but decided advance, I have purposely quoted the first edition of each work; for the views first promulgated by Bouillaud and Hope, for instance, were very much modified in subsequent editions, and the student loses very much the sensation of gradual progress if he reads only the last edition of either. In this brief survey of the progress of cardiac diagnosis I have purposely restricted myself to practical treatises on diseases of the heart. To extend the survey wider would be to give an entire bibliography of the subject, which seems to me quite unnecessary. I may, however, add that the only two modern works on percussion and auscultation

<sup>1</sup> *The Diseases of the Heart and the Aorta*, Dublin, 1854.

<sup>2</sup> *Lehrbuch der Herzkrankheiten*, Leipzig, 1868.



which contain important novelties in the practical application or the scientific explanation of these arts are those of Skoda<sup>1</sup> and of Paul Niemeyer<sup>2</sup>; and I may also mention the work of Allan Burns<sup>3</sup> as containing some important observations, especially in regard to mitral stenosis and epigastric pulsation; the paper by Billing,<sup>4</sup> on the sounds of the heart, as containing the first promulgation of views in regard to their production, which have since been adopted, and upon which much of modern diagnosis hangs, and in this respect Rouanet's<sup>5</sup> paper is also of importance; also Corrigan's<sup>6</sup> important paper on aortic regurgitation; and the papers by Fauvel,<sup>7</sup> Gairdner,<sup>8</sup> Hilton Fagge,<sup>9</sup> and Galabin<sup>10</sup> upon mitral stenosis.

In regard to the treatment of cardiac disease, it is only in recent times that any important advance has been made. Senac says: "A mesure qu'on pénètre dans les maladies du Cœur la médecine paraît plus stérile; que peut-on espérer des médicaments, par exemple, dans les dilatations du Cœur?"<sup>11</sup> But to this doleful query modern medicine has given a most triumphant reply, and can truly say that there are few diseases more amenable to treatment than moderate dilatation of the heart, while every form of cardiac disease is susceptible of a degree of relief which but a few years ago was

<sup>1</sup> *Abhandlung über Perkussion und Auskultation*, Wien, 1839.

<sup>2</sup> *Theoretischen und klinischen Handbuch der Perkussion und Auskultation*, Erlangen, 1870.

<sup>3</sup> *On some Diseases of the Heart*, Edinburgh, 1809.

<sup>4</sup> *Lancet*, May 1832.

<sup>5</sup> *Journal Hebdomadaire*, No. 97.

<sup>6</sup> *Edinburgh Medical and Surgical Journal*, vol. xxxvii. 1832.

<sup>7</sup> "Mémoire sur les signes Stéthoscopiques du Rétrécissement de l'orifice auriculo-ventriculaire gauche du Cœur," *Archives Générales de Médecine*, tome i., 1843.

<sup>8</sup> *Edinburgh Medical Journal*, November 1861.

<sup>9</sup> *Guy's Hospital Reports*, third series, vol. xvi.

<sup>10</sup> *Ibid.* vol. xx.<sup>1</sup>

<sup>11</sup> *Op. cit.* vol. ii. livre iv. chap. iv. p. 328.

wholly undreamed of; but for a further consideration of this I must refer to the body of this work generally, and particularly to Lectures iii., vi., vii., xii., and xiv.; and I believe that a comparison of these chapters with even the latest works on cardiac therapeutics will show that a very considerable advance in this respect has been made in quite recent times, and that this advance is undoubtedly due, or at all events owes much of its precision, to the researches of physiologists. I regret I can give no definite statistical information as to the prevalence of the various forms of disease of the heart, because my own time and attention have been otherwise occupied, and I find that those statistics, accumulated for me by my various resident physicians, are not of much practical value. During the eight years I have been connected with the Royal Infirmary here, I have had under my care in its wards considerably over 2000 cases of general disease, exclusive of cases of continued and eruptive fevers. Some of the ward journals have gone amissing, but I have records of 1968 cases, of whom 200 were cases of cardiac disease. Of these 67 are recorded under the head of aortic regurgitation alone, and 18 under that of aortic and mitral disease; there were therefore 85 in whom aortic regurgitation was the most important lesion; 77 cases are recorded under the head of mitral stenosis, and 48 simply under that of mitral disease; of these, therefore, a pre-systolic murmur must have constituted the predominant sign in the first series, and a systolic murmur in the second—125 cases in all of mitral disease, and 213 in all of aortic and mitral disease, exclusive of cases of simple dilatation, of dilated hypertrophy, of tricuspid regurgitation or obstruction, or of pulmonary obstruction. These statistics are obviously of no value except as affording an approximate indication of

the numbers of cardiac cases coming annually under my observation in the Infirmary alone.

I have also to apologise for the large number of cases recorded in the chapter on the treatment of aortic aneurysm, as well as for their fulness of detail, but the subject appeared to me of so much importance as imperatively to call for full information. I may add that I have had under my care in the Infirmary during the last eight years over 30 cases of aortic aneurysm, of whom 31 were cases of thoracic aneurysm, 23 of them males and 8 females, and 5 were cases of abdominal aneurysm (aortic), all males.

In conclusion, I may say that no one can be more conscious of the many shortcomings of this work than I am myself, or, I may add, more desirous that any opinions advanced in it should only be received in so far as they shall be found to be consistent with the truths of those sciences upon which the successful practice of our profession is based.

EDINBURGH, *December* 1875.



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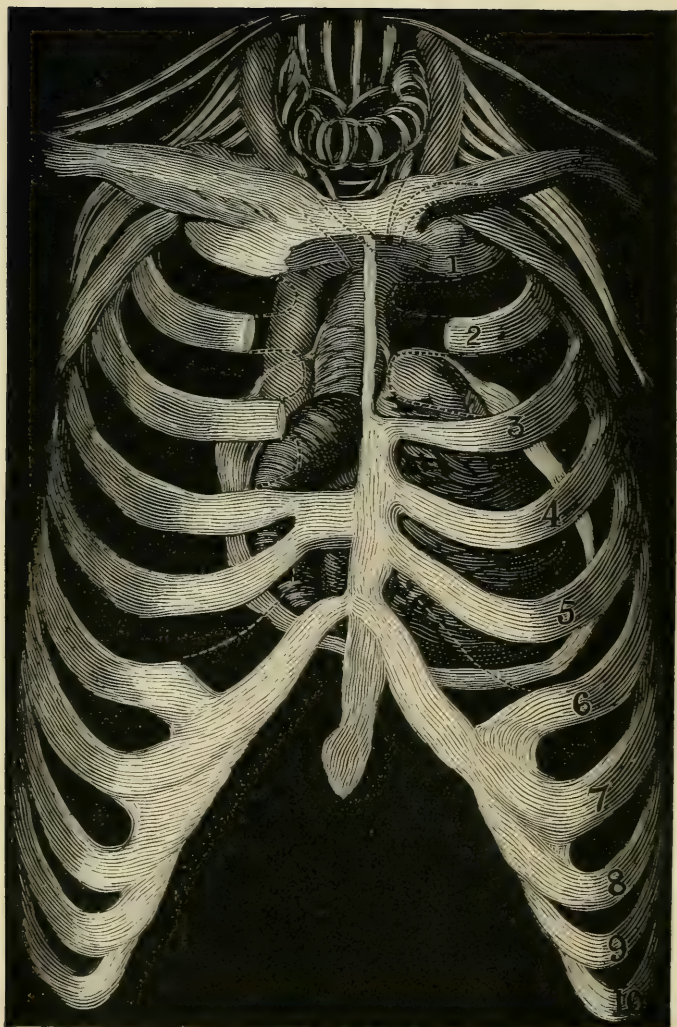
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Slightly altered from Sibson (*Medical Anatomy*, plate XIX.)



## LECTURE I

### ON THE DIAGNOSIS OF CARDIAC DISEASE GENERALLY, WITH SPECIAL REFERENCE TO THE SYMPTOMS AND PHYSICAL SIGNS

IN gathering together the prominent facts in relation to the circulatory system, few things are more remarkable than the apparent irrelevance of the symptoms complained of. Thus if a patient complains of palpitation, irregular action, or of his heart generally, you may, if he is young, assure him without much fear of being wrong that his heart is all right, and that he is only weak, nervous, and probably dyspeptic. In those past middle life the case is different, in them every symptom is of importance, however trifling it may seem.<sup>1</sup>

We continually meet with cases in which serious valvular lesion has existed for many years altogether unnoticed by the patient, notwithstanding that he has been leading an active and laborious life. Such a lesion is said to be mute; it has been perfectly compensated, and it never asserts itself as a disease until the compensation has been ruptured by accident, or by the gradual advance of those organic changes associated with it, either as cause or effect. In the one case the ruptured compensation is reparable, and comparative health may be restored; in the other, it is irreparable,

<sup>1</sup> Vide *The Senile Heart*, by George W. Balfour, M.D. Adam and Charles Black: London, 1894.

though judicious treatment may prolong life and postpone the inevitable end.

A truly cardiac patient—one suffering from actual disease of the heart—as a rule comes to you complaining not of his heart, but of one or other of the secondary results of his lesion. He complains of breathlessness or of dropsy, either or both of which may result from that lesion if it be insufficiently compensated, or if the compensation be ruptured.

If breathlessness is complained of, you will find that cardiac breathlessness presents certain peculiarities by which it is readily distinguished from pulmonary breathlessness. The most striking of these differences, and one which at once attracts attention, is the perfect tranquillity of the breathing while the patient is at rest, while the slightest exertion at once produces so anxious a desire for more air as can be expressed by no fitter term than the *air-hunger* of the Germans. The amount of lesion is not to be measured by this breathlessness, but its seriousness, as dependent upon the degree in which the compensation is ruptured, may certainly be so. Thus a patient may only puff considerably on going up a hill or ascending a stair, or his shortness of breath may be so great as speedily to compel him to call a halt on attempting either of these feats; or it may be so extreme as to prove distressing on making such a perfectly trifling exertion as sitting up or turning in bed. But withal there is no true dyspnoea, no difficult breathing properly so called; there is no obstruction either to inspiration or to expiration; there may even be no curtailment of the air-space in the lungs from any cause whatever; the breathing while the patient is at rest is perfectly quiet and natural; yet such is the difficulty—from cardiac causes—of getting the blood aerated, that the slightest exertion produces such a gasping inquietude as is extremely characteristic. This is one form of cardiac asthma, as it is termed; but there is another in which the breathlessness, though not dependent on exertion, is yet equally independent of pulmonary lesion. This latter

variety may come on at any time, but it is, perhaps, most usual at night, and wakes the patient from his first sleep gasping and alarmed; he has palpitation, occasional pain (angina), almost always irregular action of the heart, which is always feeble; now and then the patient is sick, and sometimes he vomits a mouthful or two. This form of cardiac asthma is mostly senile in character, and associated with muscular degeneration rather than with valvular lesion. It is caused by some sudden rise of blood-pressure of reflex origin, and it is often the beginning of the end to those affected—the first intimation that “the pitcher is broken at the fountain,” and that death has already seized upon the very citadel of life.

Such patients, however, never come to see you—you are always sent for to see them; and I only mention this affection now as an illustration of the fact that exertion is not always necessary to produce cardiac breathlessness, and that the panting and the absence of true pulmonary distress are always characteristic.

Breathlessness depends upon imperfect aeration of the blood, and in the absence of any pulmonary lesion it may depend upon some lesion of the heart or of the blood itself. Thus, though a patient presents all the characteristics of cardiac breathlessness, we must not set him down as certainly labouring under cardiac disease, he may be only anæmic or spanæmic. The heart may be right enough and only the red corpuscles deficient; but as anæmia and heart disease frequently coexist, the assured presence of the former, evinced by the pallor of the lips, gums, etc., does not exclude the latter. Breathlessness having the characteristic symptoms described, makes us certain we have to do with a hæmic or a cardiac lesion; which it is we must determine by further inquiries.

Dropsy depending upon cardiac disease always<sup>1</sup> begins across the instep, and gradually fills up the lower extremities,

<sup>1</sup> “Almost invariably,” says Walshe, “any exception being excessively rare.”—*Diseases of the Heart*, 3rd edition (London, 1862), p. 302.

the face and upper parts of the body remaining free. But as the œdema of simple debility begins in a similar position and follows a similar course, dropsy in this position can only be accepted as a hint that possibly the heart may be affected, and not as a proof that it is.

*When a patient, then, breathes easily when at rest but complains of breathlessness on exertion, or has swelled feet with or even without marked breathlessness, we suspect his heart, and we confirm or set aside this suspicion by further inquiry.*

In further examining the condition of the patient we begin by feeling both radial pulses simultaneously, noting whether the arteries are firmer or more tortuous than usual (atheroma). If there is a marked difference in size between the two radial arteries, we feel both brachials simultaneously; if these be equal, the difference between the two radials is probably due to irregular distribution, which must be looked for. If the brachials also differ in size, there is probably some abnormal physical cause for this irregularity—possibly an aneurysm. Should the radial pulses be equal and regular, but small and feeble, we raise the wrist to a level with the top of the head, if the patient be sitting or standing; if he is lying, we raise the arm to its full length perpendicularly to the body. Should the pulse on this procedure become extinguished, or nearly so, the patient is anæmic, and probably anæmia is the sole complaint. But as we can never rely upon one symptom for a diagnosis, we merely note the fact observed as one step in our progress. Should the pulse, after elevation of the arm, remain distinct though small and feeble, any cardiac disease present affects the mitral orifice. Irregularity, if present, helps to confirm this suspicion; and extreme irregularity with a small pulse points to the probability of this mitral affection being stenosis rather than simple insufficiency. Should the pulse, though small, feeble, and irregular, not only remain distinct after elevation of the arm but apparently become more so, the impulse seeming more forcible from the sudden and complete collapse of the artery

which immediately follows, then we know we have to do with a double lesion, with regurgitation through the aortic as well as through the mitral opening. This form of pulse cannot be relied upon unless the collapse is distinct; unfortunately it is seldom well marked, and is not, therefore, easily detected. The ordinary water-hammer or Corrigan's pulse<sup>1</sup> of simple aortic regurgitation is readily distinguished by the peculiar sensation conveyed to the finger, a sensation which is generally well marked, and frequently so exaggerated by elevation of the arm as to be wholly unmistakable, and sometimes almost painful.

*While certain general symptoms indicate with greater or less probability the existence of cardiac disease, the examination of the pulse alone may not only confer more or less certainty on the suspicions thus aroused, but may even enable us in some degree to predicate the nature of the lesion.*

We next examine the patient by a careful INSPECTION of his thorax and neck. A normal thorax is perfectly symmetrical on both sides of the sternum. The chest walls gently rise and fall rhythmically with inspiration and expiration, and the only break in the monotony of this gentle undulation is a slight tap visible between the fifth and sixth ribs, about two inches from the left edge of the sternum.<sup>2</sup> This tap is the beat of the heart's apex upon the inner surface of the chest wall, it is an outward thrust rather than a tap, and it averages four thrusts to each respiratory wave. Any deviation from these appearances is abnormal, though not necessarily of much importance. Alterations in the shape of the chest from rickets, scoliosis, or unilateral pulmonary disease, are only of consequence in the present connection when the pulmonary circulation is so interfered with as to induce secondary affection of the right heart, or when parts

<sup>1</sup> *Edinburgh Medical and Surgical Journal*, vol. xxxvii. (April, 1832), pp. 227, 229, where the phenomena referred to are described for the first time by the late Sir Dominic Corrigan.

<sup>2</sup> Those who have worked with female patients will appreciate the discarding of the nipple as a fixed point.



ordinarily covered get exposed, revealing pulsations that are apt to be taken for aneurysms. In many cases the precordial region is more prominent than the similar region on the right of the sternum. Of itself this is of little importance, but it is frequently associated with enlargement of the heart or with effusion into the pericardium. In ascertaining its presence we must be careful not to be misled by any rachitic bulging of the ribs, or by any unusually distinct pulsation of the heart in children or in meagre individuals. Bulgings due to arterial disease (aneurysm) invariably commence above the fourth rib, and appear as mere local tumours. Depression of the præcordial region is much less common than bulging; it occurs, but very rarely, as a result of pericarditis, following adhesion of the visceral and parietal layers of the pericardium and of the superjacent pleura; more commonly, but still rarely, there is a mere systolic recession of the præcordial region from the same cause, but this systolic recession is more usually due to atmospheric pressure during the systole of a large heart, quite apart from any pericardial adhesion. Any alteration in the position and extent of the apex beat is readily observed, and must be carefully noted for further investigation. Above the chest wall in the neck we note whether there is any undue *pulsation* in the *carotid arteries* or in the tracheal fossa; this is often found in advanced life when the vessels are atheromatous and tortuous, but if symmetrical and excessive it is invariably associated with regurgitation through the aortic valves, and coexistent dilatation and hypertrophy of the *left ventricle*. Visible *venous pulsation*, on the other hand, is invariably associated with dilatation of the *right ventricle*, and its distinctness may be accepted as a measure of the persistence and degree of that dilatation, because venous *pulsation* in this situation is never seen till long-continued congestion has destroyed the competency of the valves at the root of the jugular vein, and this does not happen till some considerable time after tricuspid regurgitation has been

established. Venous pulsation is generally best seen in the right jugular vein and is brought out by compressing the vein about the middle of the neck, the natural supply of blood from above is thus cut off, and the vein is seen to fill from below in a succession of waves synchronous with each cardiac pulsation. Simple *undulation* of the jugular vein is caused by the propagation of each systolic impulse of the right ventricle through the congested auricle and along the turgid vein, the jugular valves remaining intact. Jugular undulation thus indicates a much shorter duration of tricuspid regurgitation than is betokened by jugular pulsation. A mere flicker at the root of the jugular vein is of no importance, it is seen in most healthy people lying supine. Venous pulsation is often difficult to detect in anæmic persons, especially if they are young, as the veins are small and apt to be concealed by the subcutaneous fat, always more abundant than in older people.

Below the anterior chest wall we may observe pulsation in the epigastric region from various causes. Epigastric pulsation is sometimes associated with absence of the apex beat from its normal position, and is then to be looked upon as one form of its displacement. When the right ventricle dilates it pushes the left ventricle back from the chest wall and communicates its own pulsation to the lower part of the sternum and to the liver, which may then be seen to pulsate in the *scrobiculus cordis*. It may be doubted if such pulsation is ever seen in a perfectly normal condition of the heart and neighbouring organs, but it is certainly often observed when no actual disease of the heart exists. Whenever pulmonary congestion is present, even as the result of such simple and temporary causes as strenuous exertion or bronchial catarrh, some degree of dilatation of the right ventricle always exists, and may give rise to epigastric pulsation. This is more easily seen when circumstances favour the transmission of the cardiac impulse to the abdominal wall, as when there is effusion into the pericardium or

enlargement of the liver, the last common enough in connection with a dilated right ventricle. The systolic impulse of regurgitation through the *vena cava inferior* in such cases is sometimes great enough to communicate a distinctly expansile pulsation to the whole liver, a pulsation not confined to the *scrobiculus cordis*, but perceptible over the whole right hypochondriac region.<sup>1</sup> A large aneurysm resting above the right lobe of the liver sometimes communicates pulsation to that organ of so extensive a character<sup>2</sup> as to simulate expansion. The pulsation of the abdominal aorta is sometimes distinctly visible in the epigastric region simply from thinning of the abdominal walls from emaciation, or the aortic pulsation may be communicated to the surface by some overlying tumour, and in this case the pulsation may be limited in its vertical direction and may even extend transversely. Now and then we have a saccular aneurysm projecting from the anterior surface of the abdominal aorta, and perhaps more frequently we have a mere neurotic throbbing strictly limited to the abdominal aorta itself. In certain rare cases a pulsatory movement is communicated to the epigastrium by the heart's apex pulling upwards an adherent pericardium, diaphragm, and liver during the

<sup>1</sup> Allan Burns was the first to describe this form of hepatic pulsation in his *Observations on Some of the Most Frequent and Important Diseases of the Heart* (Edinburgh, 1833). At p. 265 he quotes a case from Senac, in which direct pulsation was communicated to the epigastrium by a *vena cava inferior* the size of a man's arm. At p. 266 he also mentions a case in which epigastric pulsation was produced by repercussion from solidified lungs.

<sup>2</sup> Extract from a letter from a late physician, who suffered from an aneurysm of the thoracic aorta projecting through the sternum: "I am satisfied that your view of the origin of the liver pulsations from the aneurysm mainly is the correct one, however discouraging to myself that may be. But I have this to say on the more cheerful side, that if an *enlarged* liver has had nothing to do with them (as Dr. — says), and which by decreasing in bulk has caused them to be less felt, the very great decrease in them which has occurred since I began the iodide is very favourable to the conclusion that under its action the sac must have contracted considerably. Early in February, before I had reason to suspect any increase of the liver, the pulsations had the effect of expanding the opposite sides of the hypochondria at each beat, an effect which is not now perceptible, though I am supposing the liver to be larger, in which, however, I may be mistaken." Dated 3rd May 1870.

ventricular systole.<sup>1</sup> This movement is exactly the reverse of that in ordinary epigastric pulsation, there is a systolic depression instead of a systolic outward thrust. As so extensive an adhesion is, as a rule, the result of severe inflammation affecting the whole surface of the heart, it happens in such cases that the cardiac pulsation is apt to appear as a mere undulatory movement, and it may be difficult to time any particular part of this movement so as to determine whether it is systolic or diastolic, especially if the heart's action is at all rapid (over ninety beats per minute). To this end we must attach a short bristle carrying a tiny paper flag, by means of a pellet of beeswax to the skin over the apex of the heart, and we place a similar flag over any other point in regard to whose time (position in the cardiac cycle) there may be a doubt. The eye readily discriminates any difference in time in the movements of the flags, and with care we can generally determine whether the movement in question precedes or follows the apex beat. This method of investigation is often useful in the case of pulsations above the fourth rib, which may either be auricular or aneurysmal (aortic); in the former case the pulsation may precede the apex beat, in the latter it must either be synchronous or follow it. In certain rare cases we get intrathoracic pulsations which originate from the heart, and yet have no connection whatever with any disease of the circulatory organs. Thus in some cases of pneumonia the hepatised lung conveys to the surface of the chest a pulsatory thrill; in certain cases of malignant disease of the lung the pulsation is even more perceptible; and in cases of purulent effusion into the pleural cavity (empyema) pulsation is often very visible.

*From INSPECTION we learn the general configuration of the chest, and any apparent deviations from the normal; the eye also notes any movements—other than the rhythmic respiratory*

<sup>1</sup> A remarkable instance of this will be found detailed on p. 214 of Copland's *Dictionary of Practical Medicine*, vol. ii.



*movements and the isolated tap of the heart's apex—that may be seen on the neck, the chest wall, or the epigastric region, but these are noted only in order that their exact nature may be fully investigated by other methods of exploration. A single glance reveals to a man of experience the probable nature of these abnormalities, but a student must painfully inquire into every detail.*

On placing the hand over the cardiac area, we can, by PALPATION, if the chest wall is thin, readily perceive the alternate movements of systole and diastole over both the auricular and ventricular areas. A forcible ventricular impulse is a probable indication of hypertrophy ; but a feeble impulse is not to be looked upon as any certain indication of atrophy, predominant dilatation, or even of debility of the heart (myocardium) from any cause. A feeble impulse may mean any of these, or it may be caused by considerable effusion into the pericardium, by pulmonary emphysema, excess of fat overlying the heart (*adipositas cordis*), or merely by increased density of the thoracic wall. If we cause the patient to sit up and lean well forwards the cardiac impulse is more readily felt in every case, and specially so when the displacement from the chest wall has been due to fluid in the pericardium. The greater or less distinctness with which any pulsation is to be felt, and its exact position, must be carefully noted in every case. Forcible pulsation above the fourth rib, and within the cardiac area (*vide* frontispiece), may be aneurysmal (arterial) in character, but it may also depend upon dilatation and hypertrophy of the auricle, especially when it occurs on the left of the sternum. Forcible pulsation below the fourth rib and to the left of the cardiac area is associated with depression of the apex beat below the normal (fifth interspace), and indicates dilatation and hypertrophy of the left ventricle. Forcible pulsation beneath the lower end of the sternum, with displacement of the apex beat to the right of its normal position (two inches from the left edge of the sternum), indicates dilatation and hypertrophy



of the right ventricle, the increased size of the ventricle being to some extent measurable by the force of the impulse and the amount of epigastric pulsation present. When the whole heart is dilated and hypertrophied, a more or less violent shock may be felt over the entire cardiac area; and when the size of the heart is considerable this shock may be double—the first forcible and systolic, the second less forcible and diastolic, the result of the rebound of the enlarged heart from the posterior thoracic walls.

In the normal state inclination of the patient to the left displaces the apex beat to the same side, sometimes even a little farther to the left than a point equidistant between its normal position and the axillary line. If we incline the patient to the right, the apex beat grows faint or disappears, the right ventricle being felt to beat distinctly beneath the lower end of the sternum or even in the epigastric region. When fluid is effused into the pericardium during an attack of pericarditis the heart's apex falls back from the chest wall, and it is the comparatively immobile base of the heart that is felt beating, often as high as the fourth interspace and slightly towards the left of the normal position of the apex. The apex in such a case is said to be displaced upwards. When there is much fluid in the right pleura the heart is displaced towards the left; when the effusion is into the left pleural cavity the heart is turned round and forced over to the right side, sometimes as high as the fourth interspace.<sup>1</sup> In hypertrophy with dilatation of the left ventricle, the apex passes downwards and to the left. In a similar condition of the right ventricle, the apex disappears from its normal position and a diffuse impulse is found at the lower end of the sternum.

When from any cause the left lung is retracted from the base of the heart, the pulsation of the pulmonary artery may be felt between the second and third ribs, close to the left edge of the sternum; and even the click of the semilunar

<sup>1</sup> *Collected Works of Francis Sibson, M.D.* (London, 1881), vol. iii. p. 221.

valves may be perceived, because sound is caused by vibrations which may be felt as well as heard when the conducting medium is suitable. Hence the palpating hand can readily feel the friction produced by the rubbing together of the two layers of the pericardium roughened by adherent lymph, and may even recognise the vibrations of valvular murmurs. When these murmurs are rough the term of purring thrill—*fremissement cataire*—has been applied to the vibrations felt, because the sensation produced resembles that perceived on placing the hand on the body of a cat in the act of purring. Abnormal pulsations along the course of the thoracic aorta are most readily perceived in connection with the ascending and transverse portions of that vessel, they may be due to displacement and uncovering of the vessel, as occasionally happens in deformed and rickety chests, or they may be caused by aneurysmal bulgings. They are to be sought for in the tracheal fossa, and between the ribs along the course of the aorta, especially during expiration, when such pulsations, if faint, are most readily felt. Abnormal pulsation may also be felt along the course of the descending aorta, close to the left side of the spinal column posteriorly, but only when the aneurysm has attained some considerable size.

*From this we see that a great amount of information necessary for diagnosis is to be obtained by PALPATION. The points to be specially noted are—1st, the position of any perceptible pulsation; 2nd, its extent and force; 3rd, its rhythm, whether systolic, diastolic, irregular, or intermittent; and 4th, whether any vibratile sensations are perceptible over the seat of pulsation, and if so, their rhythm.*

Having registered all the information to be obtained from the inspection and palpation of the patient, we proceed to percuss him.

PERCUSSION, in medical parlance, is the art of ascertaining the size and shape of the internal organs of the body by percussing, or tapping, the overlying surface. Percussion may be made by tapping with the tips of the fingers directly

on the surface of the body: this is called immediate percussion. Or we may employ a hammer, or plessor as it is called, to strike with, and a finger or piece of ivory to receive the blow, this interposed recipient of the blow being called a pleximeter: this mode of percussion is called mediate. When we use the fingers directly, we not only hear the sounds produced, but our tactile sensation enables us to recognise the density of the body struck. On the other hand, the use of a hammer and pleximeter enables us to produce a louder sound, and is thus advantageous in teaching a class. When we strike or percuss any body sufficiently tense to be thrown into independent vibrations, it gives out a note of its own by which it is at once recognised. But if it be not tense enough to be thrown into independent vibrations, it simply gives out no note at all. When bodies vibrate on being struck in the open air they produce a distinctly recognisable tone, but devoid of fulness or duration; but if the same object is set in vibration over a resonance box the tone at once acquires a remarkable fulness and persistence, from the air within the box being thrown into consonating vibrations which strengthen and reinforce the original tone. Resonance, consonance, or reinforcement happens when the vibrations of the object struck synchronise with those into which the air within the box is thrown. This can only happen when the depth of the box—length of the air-column contained—exactly equals one-fourth of the wave-length of the original tone. That is to say, a resonance box—or rather the air within it—consonates with and reinforces only that tone one-fourth of whose wave-length exactly equals its depth, and is silent to all others. The thorax is a large resonance box within which lie the heart and large blood-vessels; these organs, in their normal condition, contain no air, and are never sufficiently tense to be themselves thrown into sonorous vibrations by percussion; when struck, therefore, mediately or immediately, they give forth no sound at all, they are said to be dull on percussion. In their normal position and condition the heart and large

blood-vessels are surrounded on three sides by the lungs, which normally are filled with air and therefore resonant; on the fourth side the heart rests upon the liver, like itself a dull, non-resonant body, from which we can separate it more or less perfectly by measures presently to be described.

On percussing the anterior chest wall, the veriest tyro can

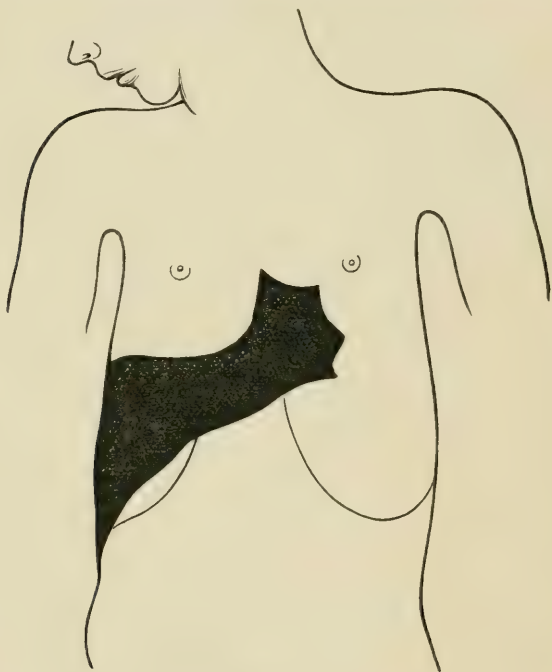


FIG. 1.

at once distinguish a dull area at the lower part of the right chest, and extending into the left chest as a more or less triangular patch below and to the right of the nipple (Fig. 1<sup>1</sup>).

<sup>1</sup> This figure is a diagrammatic representation of Fig. 4—organs healthy—of “Illustrations of the Morbid Anatomy of the Organs of the Chest,” by Francis Sibson, published in the twelfth volume of the *Transactions of the Provincial Medical and Surgical Association*. It has been selected because the area of cardiac dullness approaches more nearly in configuration the ordinary conventional idea of such dullness than that of any of the other figures in the same work. How greatly this may vary may be seen by a reference to those other figures representing bodies with healthy organs.

On the right side this dulness is entirely due to the liver. On the left side it is partly due to the liver and partly to the heart; to separate the heart dulness from that of the liver is impossible, but we practically attain this end by feeling for the apex beat and tracing a straight line from it to the upper part of the liver dulness on the right side; all the dulness above this line and to the left of the sternum may be considered as due to the heart alone. This area of marked, easily detected, or as it is called, superficial dulness is an exact measure of that part of the heart in immediate contact with the chest wall. It is no indication of the size of the heart generally, but only of the extent of that organ uncovered by the lung, and as this varies according to the size of the heart itself, and the degree of expansion of the lung, the positive information conveyed has but a limited value. To ascertain accurately the size of the heart we must map out the entire area of cardiac dulness, both superficial and deep, to use for the nonce those anatomical expressions which are so constantly applied to acoustic phenomena, in spite of Auenbrugger's vigorous protest that acoustic phenomena only reveal physical conditions, and from these the anatomical relationship of the organs can only be deduced by a process of reasoning.<sup>1</sup>

The heart and large blood-vessels occupy the centre of the thorax, extending from the upper border of the third rib to the lower end of the sternum, and almost entirely fill up the space between the sternum and the vertebræ. In this position the ventricles encroach more upon the left lung, and the auricles upon the right one, the whole of the right cavities lying in front and slightly to the right of the left cavities. The axis of the right ventricle in relation to the pulmonary artery is almost vertical, the broadest part of the ventricle being that most distant from the pulmonary orifice. The

<sup>1</sup> *Inventum Novum*, 17, 18, scholia. Auenbrugger was the first to apply the art of percussion to the discrimination of diseases of the chest. The earliest edition of his work was published at Vienna in 1761.



axis of the left ventricle is almost horizontal in relation to the aorta, the narrowest part of the ventricle being that most distant from the aortic orifice. From this peculiar formation of the ventricles, and from the anterior position of the right one, it happens that the pulmonary artery rises in front and to the left of the aorta.

From the formation and position of the heart, it is obvious that, though we can and may percuss out the whole of the cardiac dulness, this is quite unnecessary; it is only of importance to ascertain the limits of this dulness vertically and transversely. Increase of the vertical dulness, in the parasternal line, rarely indicates any alteration in the size of the heart itself, but is usually due to effusion into the pericardium or to hepatic enlargement. Dulness from the former cause lies above the third rib, and from the latter, as a rule, below the sixth rib; these conditions have to be noted for further investigation. A simple change of position of the heart, which may arise from various causes, is indicated by a transference of the normal dulness upwards or downwards, without any change in its extent. Except in certain abnormal states the apex beat is, from the formation of the heart, that part of it which lies farthest to the left, and being as a rule perceptible to the touch, it only requires to be percussed out in those exceptional circumstances in which the apex beats beneath a rib, and not in an interspace. The right auricle is that part of the heart which lies farthest to the right, and being extremely dilatable, and readily influenced by any obstacle to the onward flow of the blood, the transverse dulness at the level of the fourth rib comes to be an important indication of enlargement of the heart, chiefly in its auricular region, due to some obstruction to that onward flow.

The chief points in regard to the state of the heart, therefore, upon which percussion supplies us with information, are—

*The presence of dulness above the fourth rib, in the parasternal line, which, as a rule, indicates effusion into the pericardium.*



*And, secondly, marked increase of the transverse dulness at the level of the fourth rib, which indicates obstruction to the circulation of the blood.*

If, along with this increase of the transverse dulness, the apex beat is displaced downwards and to the left, this obstruction is most probably of aortic origin, and has primarily influenced the left ventricle. But if the apex beat is displaced to the left but not downwards, the obstruction is either of mitral or of pulmonary origin.

In further endeavouring to map out the exact size of the cardiac dulness, we must not forget that the heart is an organ which is perpetually changing in size, and in its relation to the surface, and that it is surrounded on three sides by the lungs, organs which are themselves continually undergoing changes in size, dependent on respiration. So that but little experience is needed to assure us that even the normal heart undergoes many rapid alterations in the extent of its percussion dulness.<sup>1</sup> Thus, though percussion is a valuable means of determining the superficial area of the heart, it is only after an average of several observations has been obtained that we can arrive at even an approximate conclusion as to that, while any inference as to the relative condition of the cardiac walls and cavities (state of hypertrophy or dilatation) can only be made after a careful collation of other correlative data. In percussing the region of the heart, we must remember that, so far as the three sides surrounded by the lungs are concerned, the passage of the percussion note from clear to dull is not abrupt, but transitional, the heart on these three sides being covered by a gradually decreasing layer of lung. Practically we find that the percussion note ranges from above downwards, in the parasternal line, from

<sup>1</sup> *Vide* "Perkussionsverhältnisse am normalen Herzen," von Dr. M. Heitler, *Wiener klinische Wochenschrift* (1890), Nos. 41 and 42. In this paper the periodical variation of the cardiac dulness is shown to be entirely independent of any change in the lungs, as increased dulness occurs normally three or four times a minute, and coincides with the inspiration as often as with the expiration. These variations are sometimes very considerable.

clear and full just above the upper border of the third rib to perfectly dull somewhere below the lower border of the fourth rib,—the note becoming gradually less full but still remaining perfectly clear until the upper limit of superficial dulness is attained.

A perfectly dull sound is produced by percussing over a mass of solid flesh, such as the thigh, and indicates that

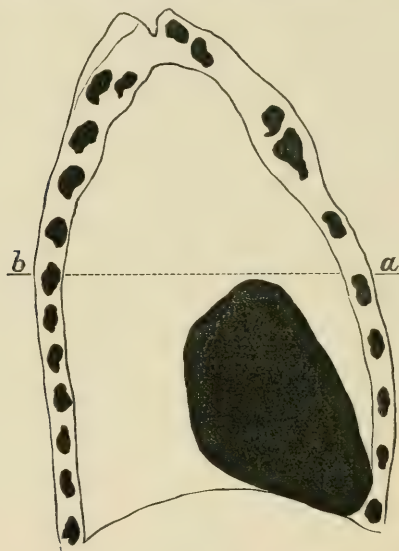


FIG. 2.

there is neither air nor any gaseous body beneath the part percussed.

A glance at the accompanying diagram (Fig. 2<sup>1</sup>) shows that, in the normal condition, a mass of air-containing lung

<sup>1</sup> This figure is a diagrammatic representation of Fig. 2, table 5, of Fasciculus 2A of Pirogoff's *Anatomia Topographica Sectionibus per Corpus Humanum congelatum triplici ductus illustrata* (Atlas, Petrop., 1859). The section passes at the upper part through the left sterno-clavicular articulation, at three Paris lines from the internal margin of the head of the left clavicle. The lowest rib anteriorly is the seventh, cut through ten lines from the sternum; posteriorly, it is the twelfth rib, cut through nine lines from its vertebral extremity. In this section the lung descends lower than is usual, so that perfect dullness would not commence till the lower edge of the fifth rib, instead of that of the fourth rib, as is customary.

occupies the whole of the thoracic cavity above the third rib. Beneath the third rib this air-space is seen to be gradually encroached upon by the heart as it approaches the anterior chest wall, with which it comes in contact about the lower border of the fourth rib. In percussing along the parasternal line, the note elicited above the third rib is full, that is, of a duration and volume proportionate to the size of the air-space (resonance box). The sound is also clear in proportion to the elasticity of the chest wall, and the readiness with which this is thrown into vibration. Beneath the third rib the sound elicited on percussion gradually gets less full until it is lost in the cardiac dulness, and a glance at the diagram shows the reason for this in the gradual diminution of the air-space beneath the part percussed. Skoda has applied the term *leer*, or empty, to a sound thus lessened in volume by filling up the air-space from below. A percussion sound that becomes gradually empty may remain perfectly clear to the last, provided the chest wall is elastic, until the perfectly empty sound merges in its equivalent, the perfectly dull sound of the heart below the fourth rib. Should the chest wall be less elastic, the sound elicited on percussion is muffled or dulled just in proportion to the decreased capacity of the medium percussed for undergoing sonorous vibrations. A percussion sound becomes emptier, less full, the duller or more muffled it becomes; but it by no means necessarily grows duller as it gets emptier, because a sound may be very empty and yet perfectly clear. The terms full and empty are thus synonymous with a greater or less intensity of sound, the quality of intensity depending on the volume of sound that reaches the ear.

Acoustic phenomena are generally spoken of in relation to their intensity, pitch, and clang or *timbre*. By many observers the phenomena just referred to *intensity*, and spoken of as full or empty sounds, have been attributed to pitch, and regarded as low or high sounds; and, indeed, an emptying of the sound is necessarily accompanied by a

heightening of its pitch. The length of a column of air which most perfectly resounds to the vibrations of a tuning-fork is exactly equal to one-fourth of the length of the sound-wave produced by the fork, and the vibrations constituting this sound-wave increase in rapidity as the wave itself decreases in length; the more rapid the vibrations, and the shorter the sound-wave, the higher is the pitch of the sound produced, as we learn from experiments on the syren. Hence the shallower any air-space, the higher the note with which it most perfectly resonates; a percussion note becoming emptier becomes, therefore, also higher in pitch. Not that there is any alteration in the general rate at which any membrane vibrates because the resonance box over which it is stretched is shallow, but because the air-space beneath the part percussed resonates only with certain of the vibrations, and raises them only to the dignity of an audible tone.

Pitch is that quality of sound most readily recognised by the average ear; it is also a strictly acoustic term, which dulness is not. It is therefore sufficient for the student to note the alterations in pitch obtained on percussing the chest wall, assured that whenever the pitch of the percussion note rises, the air-space beneath the part percussed is proportionately diminished. A knowledge of anatomy will enable the student to recognise whether his phonographic map of the chest wall is normal, or whether he will require to call pathology to his aid to explain the abnormal alteration.

*Clang* is produced by the mingling of the overtones or harmonics of the vibrating body itself with its fundamental note, and therefore varies with the nature of the structure thrown into sonorous vibrations by percussion—varies, therefore, as we percuss the intercostal tissues, the ribs, or the sternum; varies even with the nature of the plessor or of the pleximeter employed. Nay, more, clang mingled with resonance has its influence in changing the character of the percussion note in those cases where, from emaciation and



the size of the pleximeter, the latter rests upon two ribs, leaving an air-space of varying depth beneath.

As clang varies with structure, tension, and elasticity, we must—to avoid its confusing influence in estimating the exact quality of a percussion note—be careful in percussing from above downwards to compare rib with rib, and interspace with interspace. For the same reason, in percussing transversely, we must be careful to percuss along a rib or along an interspace, and avoid shifting indefinitely from one to the other. Both pleximeter and plessor must also be as much as possible free from clang themselves, and the former must be capable of close and accurate application to either rib or interspace. In these respects no instruments are comparable to the forefinger of the left hand for a pleximeter and the first two or three fingers of the right hand as a plessor, the only drawback to their use—and it mainly applies to class teaching—is the comparatively little intensity of the sound produced. This, however, is readily overcome by the use of a Winterich hammer as a plessor; by means of this an adept can produce a note of perfectly sufficient intensity, without injury to the finger pleximeter. An unskilful percussor using such a hammer is apt not to tap smartly enough. He may hit hard enough, there is not much likelihood of failure in that respect; but he does not lift the hammer quickly enough, and he thus muffles or stops the sonorous vibrations it is his object to excite. It occasionally happens that the rise in pitch due to the right auricle is not appreciable to the right of the sternum, and under these circumstances the clang of the sternum may be so sonorous as, in percussing transversely, apparently to do away with all cardiac dulness. But, unless under very exceptional circumstances, we can remedy this by percussing from above downwards, and by eliciting the sternal note above the aorta we can readily recognise the rise in pitch due to the heart lying beneath the sternum. One great advantage of employing the fingers as plessor and pleximeter

is that we can thus readily recognise the resistance, and this sensation of resistance supplements and confirms the auditory phenomena.

In percussing the cardiac area we must always remember that the aorta does not extend like a bow across the sternum, as Piorry has figured it, but that, as anatomy teaches, it is in close contact with the heart and pulmonary artery, and can only be mapped out by percussion as a somewhat rounded projection at the base of the heart. We must also remember that, as a rule, the dulness of the innominate artery is scarcely perceptible, while that of the left carotid, as well as that of the left subclavian, is altogether imperceptible. For this the clang of the ribs over them is partly to blame; but it is, no doubt, chiefly due to what is called the inflection of sound, by which a sonorous wave embraces a non-resonant body on all sides, and may so far extinguish the dulness of a small one as to make it quite inappreciable. Hence marked dulness in these regions is usually significant of some considerable morbid alteration.

As the heart rarely rises above the level of the third rib, and if it does so, it rises, as a rule, equally on both sides, it is obvious that Piorry's line of oblique dulness, from apex to base, is of no special practical value. And from all that has been already said, it follows as a corollary that in the percussion of the heart there are only two lines of any practical importance, viz. a vertical and a transverse one. The first of these, the parasternal line, must be thrown so far to the left as to be uninfluenced by either the aorta or the pulmonary artery, and a very convenient and suitable position for it is at one inch from the left edge of the sternum. In percussing from above downwards in this position from the lower edge of the left clavicle, *we have, in the normal condition, first of all the clear, full sound of the lung, low in pitch and of considerable duration, down to the upper border of the third rib. From the third rib downwards the percussion sound gradually rises in pitch and shortens in duration, becoming, as it is*



*called, gradually emptier, but still clear down to the lower border of the fourth, or the upper border of the fifth, rib; beneath this there is perfect dulness till we reach the tympanitic clang of the stomach or intestine.*

Any deviation from this normal gradation of sound in the parasternal line is abnormal, and its explanation must be sought for by other methods of investigation. In like manner we must carefully map out the transverse dulness in the line of the fourth rib, which is that mainly affected by any distention of the right auricle. In doing this we must percuss from without inwards on each side—that is to say, from the lung to the heart, as in that way alone can we appreciate the gradual rise in pitch, and also eliminate the clang of the sternum.

The accompanying diagram (Fig. 3<sup>1</sup>) exhibits the percussion dulness of the normal cardiac area, with the lines of practical importance (vertical *a* to *b*, transverse *c* to *d*) specially indicated. As the heart invariably rests upon the diaphragm, unless separated from it by fluid, it is never an easy matter to separate the lower part of the heart from the liver. Any change of pitch between the cardiac and hepatic dulness is inappreciable; the only possible change arises from the upward conduction of the tympanitic note of the stomach or intestines, and, of course, is not always present. We can only separate the heart from the liver approximately, by ascertaining the position of the apex beat upon the left side, and the highest point of the hepatic dulness on the right side, and joining the two by a straight line. This line of separation can never be absolutely accurate, but it is sufficiently so for all practical purposes.

It is sometimes of importance to map out the aortic dulness, and this is by no means a difficult task if set about in the right way. As already observed, it is impossible to separate the aortic dulness from that of the heart; the only

<sup>1</sup> This diagram is taken from the same subject as Fig. 1. Percussion is to be made in the direction of the arrows.

part in which these organs are not in contact being occupied by the pulmonary artery, and the percussion note of this cannot be differentiated from that of the aorta. The normal aortic dulness is indicated in the diagram (Fig. 3) as a more or less rounded area of dulness rising out of, and inseparable from that of the heart, but recognised as aortic from its position above the upper limit of cardiac dulness in the

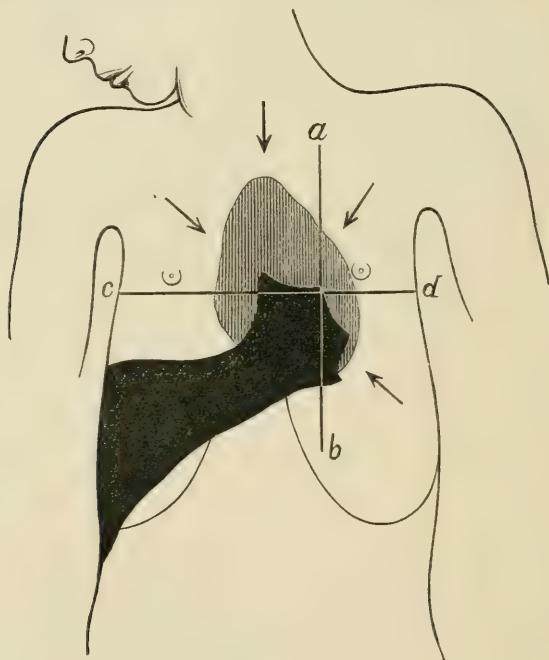


FIG. 3.

parasternal line *a* to *b*. Increase of this dulness in any direction is probably due to morbid enlargement of the aorta; but the differential diagnosis of these abnormal extensions of the aortic dulness depends upon the collation of a variety of physical facts, of which percussion dulness is but one.

Absence or diminution of the præcordial dulness is occasionally, though rarely, caused by the presence of air or

gas within the pericardium ; less seldom it is due to atrophy of the heart ; and much more frequently it is brought about by an increase of the area of pulmonary resonance, due to emphysematous expansion of the lung.

Increase of the area of cardiac dulness is a more common phenomenon. Increased dulness in the cardiac area may depend upon the presence of fluid in the pericardium, and in this case the dulness is pyramidal with the base downwards, and the cardiac pulsation is annulled, enfeebled, or displaced. Or the increased dulness may depend upon enlargement of the heart itself ; in this case the pyramidal shape of the dulness is less marked, but the broadest part is always above. When, in enlargement of the heart, hypertrophy predominates, the cardiac shock is increased ; should, on the other hand, dilatation be most marked, this shock may be almost annulled. We must never forget that these conditions may be variously modified, and to make our diagnosis correct, we must base it not upon one or two facts only, but upon all the phenomena which can be ascertained in regard to the physical condition of the heart.

AUSCULTATION is the next method we employ to ascertain the physical condition of the heart ; by this we understand the method of determining the perfection, or imperfection, of the cardiac mechanism, from the character of the sounds produced by the passage of the blood through the heart. If we place our ear over the cardiac area, we recognise that the progress of the circulation is accompanied by sounds alternated with silences. If we listen over the heart of an infant, we distinguish only a uniform ticking, in which the sounds are alike in intensity, and the silences similar in duration. But while listening to the hearts of older individuals, we become conscious that as adult life is approached these sounds, with their intervening silences, assume a peculiar rhythm, which the ear thus educated readily appreciates. And we also discover, that while the sounds and the silences remain the same over all the cardiac area, their rhythm varies according

to the position in that area in which they are heard. Thus, in listening over the apex, we distinguish two pauses or intervals of silence—a long pause and a much shorter one. Immediately following the long pause we hear a dull, prolonged sound that terminates in the short pause or silence, and this is succeeded by a short sharp sound, immediately followed by a renewal of the long pause. In this situation—over the heart's apex—the accent is upon the prolonged or first sound, as it is called, that follows the long pause, making what is known in prosody as a trochee, “—.” On the other hand, we find on listening over the base of the heart that, though the relation of the sounds to the silences remains the same, the accent in this situation falls upon the second sound instead of on the first, so that following the long pause we have, instead of a trochee, an iambus “.—.”

Though the cadences of these sounds thus vary with the position in which they are heard, they can, in the normal adult, be readily enough recognised to be first and second by their relation to the periods of silence. But whenever the heart's action becomes rapid from fever, debility, or any other cause, the long pause is shortened, and the rhythm of the sounds resembles that of an infant, so that when the heart beats over ninety per minute it is almost impossible to distinguish which is the first sound and which the second. To do so we must employ a double stethoscope, and by placing one end over the apex and the other over the base a little attention will enable us to differentiate the first sound from the second, by paying due regard to the slight differences between the two sounds, and by a careful attention to the position in which each sound is most distinctly to be heard. Apart from those alterations in rhythm, due to the rate of cardiac action, the heart-sounds vary in distinctness in each individual. Experience has taught us that in thin-walled hearts with relatively large cavities (dilated), the first sound is particularly loud, clear, and distinct; while in hearts that are thick-walled (hypertrophied) the first sound is always



muffled and indistinct. The second sound is unaffected by these conditions, it varies indeed both in distinctness and intensity, but always from causes which are extra-cardiac. We cannot, of course, securely base a diagnosis of the state of the walls of the heart upon the greater or less distinctness of the first sound, but this phenomenon supplements and confirms the information we obtain in other ways.

Besides the alterations in rhythm dependent upon variations in the rate of cardiac action, and alterations in distinctness or intensity of the cardiac sounds depending upon alterations of the myocardium, dilatation, hypertrophy, or upon some extra-cardiac cause, these sounds are occasionally replaced by noises, *bruits*, or murmurs, as we call them, which are totally different from the sounds they displace, and which vary with the cause producing them.

Physiologists teach us that the first sound coincides with the contraction or systole of the ventricles, and is composed of several elements, of which the shock of the heart's apex on the chest wall, the sound that accompanies muscular action, and that produced by the closure of the auriculo-ventricular valves, are the most important. For all practical purposes the last is all-sufficient, as we find that whenever these valves are from any cause—such as dilatation of the ventricle—rendered incapable of closure, the valves themselves remaining healthy, the first sound is either partially or wholly replaced by a murmur which more or less obscures the muscular sound, and to a less extent the shock. The second sound, on the other hand, as it immediately follows the ventricular systole, must coincide with the ventricular diastole, and therefore with that moment of time when the semilunar segments of the arterial valves are closed by the recoil upon them of the blood compressed by the arterial systole. Accordingly we find that when these valves are from any cause incapable of closure, the second sound is more or less completely replaced by a murmur, according to the degree of imperfection present.

The heart, however, is a double organ, with two auriculo-ventricular openings, and two large arteries springing from it, and in the case of valvular imperfection it is of importance to determine to which heart this imperfection belongs. But it so happens that all the cardiac valves lie so close together, that a superficial area of half an inch square will include a portion of all the four sets of valves *in situ*, while an area of about one quarter of an inch will include a portion of all of them except the tricuspid.<sup>1</sup> It is obvious, therefore, that it is impossible to differentiate the sounds or murmurs produced by one valve from those produced by another, by merely listening with an ordinary stethoscope over the place of origin. By the rhythm alone we can readily distinguish a first sound from a second one; but to differentiate a right first or second sound from a left first or second sound we must take measures to separate the sounds from each other. This we do by taking advantage of the facts that sounds produced in one medium lose in intensity in passing into another, and that sounds produced in any fluid in motion are invariably transmitted in the direction of the onward current.<sup>2</sup> Hence sounds produced in any cavity of the heart are usually heard with most distinctness over that part of the thoracic wall at which the given cavity approaches the surface most closely. Thus, the only point at which the left ventricle directly impinges on the chest wall is where the apex beat is felt, and that is

<sup>1</sup> Walshe, *op. cit.* p. 6.

<sup>2</sup> Sound is reflected, inflected, and refracted like light, and is readily conveyed to almost any distance in smooth tubes. The difficulty which sound finds in passing from one medium to another, even though these should be only strata of the atmosphere of varying densities, is well indicated by the remarkable statement that battles have been lost for want of reinforcements actually waiting, within what was thought to be ear-shot, for the sound of the cannon to indicate the moment to advance; *vide* Tyndall *On Sound* (London, 1869), p. 23, etc. A most remarkable instance of acoustic opacity of the atmosphere, associated with optic lucidity, is to be found at p. 234 of the *Philosophical Transactions* for 1874, vol. clxiv. pt. 1, where it is recorded that Mr. M'Lean and General Randolph were, at a distance of one mile and a half, for two hours spectators of the battle of Gain's Farm, in which 50,000 men and 100 field-guns were engaged, without hearing a single sound. Yet in a different direction the cannonading was distinctly audible 100 miles away.



precisely the spot where the first sound, produced within the left heart by the closure of the mitral valve, is most distinctly heard ; a space of about one inch in diameter around the apex beat is therefore termed the *mitral area*. Nearly the whole of the right ventricle is uncovered by lung, and impinges directly on the lower part of the sternum ; at this part, especially along the left edge of the sternum, where it is joined by the cartilages of the fourth, fifth, and sixth ribs, the first sound, produced within the right heart by the closing of the tricuspid valve, is most clearly to be heard ; the triangular space lying over the position of the right ventricle is therefore called the *tricuspid area*. In the normal condition the two ventricles act simultaneously, and the two sounds differ so little that it is impossible to differentiate the one from the other. If from any cause, however, these sounds become irregular or are replaced by murmurs, they may be readily enough differentiated by a reference to these special areas of audition, and as a rule there is always plenty of confirmatory proof.

On the other hand, both in health and in disease there is a marked difference between the aortic and the pulmonic second sounds, and it is often important to differentiate the one from the other, and clearly to recognise the distinctive characteristics of each. The aorta and the pulmonary artery spring from the heart close to one another. The orifice of the pulmonary artery with its valve lies about the middle of the cartilage of the third rib on the left side, one half of the valve lying to the left and the other to the right of the left edge of the sternum, which divides it exactly in two (*vide* frontispiece). From its point of origin, the pulmonary artery rises to the lower edge of the second left cartilage, where it divides into its two great branches going to the right and left lungs respectively. The second left interspace close to the sternum is thus the point at which the pulmonary artery most closely approximates the chest wall, and here, or still better at the sternal end of the third left cartilage, the

pulmonary second is most distinctly to be heard. On the other hand, the aorta rises a little below, behind, and to the right of the pulmonary artery, its valve corresponding in position to the lower edge of the cartilage of the third rib on the left side, behind and to the right of the pulmonary valve. From its origin the aorta passes upwards, forwards, and to the right, till it reaches the upper border of the cartilage of the second rib on the right side, it then passes obliquely backwards and to the left, forming what is called the arch of the aorta. At the second cartilage on the right the aorta is nearest the surface of the chest, and the arterial walls as well as the blood-current coincide in conveying to this point those resonant vibrations which result from the closure of the aortic valve,—here therefore the aortic second is most distinctly to be heard. The sternal end of the second cartilage on the right side, and of the third cartilage on the left side, are the *aortic* and the *pulmonary areas* respectively. In a state of health the aortic second is ordinarily louder and more distinct than the pulmonary second, both vessels are, however, normally covered with lung, and as, from various causes, congenital or morbid, one or other may become uncovered, and thus brought nearer the surface, its corresponding sound may be heard more distinctly than its normal, and the ordinary condition may be either intensified or reversed. But this does not happen without the occurrence of other phenomena, which enable us to detect and explain the cause of the abnormality. From the close proximity of the two arteries to each other at their origin, it sometimes happens—especially when the sternum is more than usually resonant—that the loudest sound is heard in the pulmonary area instead of in the aortic area, or towards the left of the sternum instead of towards its right. But this need give us no concern, as the aortic second can always be readily separated from the pulmonary second by carrying the stethoscope to the right or left of the sternum respectively, along the line of the second intercostal space. Besides the greater

distinctness of one sound over the other dependent upon anatomical causes, we have occasional alterations of one or other of these sounds from intrinsic causes alone. The peculiar alteration of the second sound now referred to has had a variety of terms applied to it; it has been called booming, ringing, clanging, pumping, cavernous, and accentuated; and perhaps booming or accentuated are the most unobjectionable expressions we can employ to define this peculiar change in the second sound, in which the element of tension seems united with an increase of distinctness. This accentuation of the second sound may be either of aortic or pulmonary origin, and may be heard either at the second right or the third left costal cartilage. Skoda long ago pointed out that accentuation of the pulmonary second was an important aid in the diagnosis of affections of the mitral valve; but the importance of accentuation of the pulmonary second is not limited to lesions of the mitral valve, as it is constantly present in every form of cardiac lesion involving obstruction to the onward flow of the blood. It is the most persistent of all the acoustic phenomena connected with cardiac disease, and is occasionally the only thing markedly abnormal to be detected. In the absence, therefore, of any pulmonary disease capable of giving rise to congestion, persistent accentuation of the pulmonary second is to be regarded as invariably indicative of cardiac valvular lesion. The mechanism of its production is very simple. The comparatively trifling difference between the arterial and venous blood-pressures within the pulmonary circuit necessitates of course a very slight resistance to the flow through the pulmonary capillaries, in order that, in any given time, there may be through them a blood-flow equal to that which passes through the capillaries of the systemic circulation, where very different conditions prevail. From this absence of capillary resistance, as well as from the absence of vasomotor nerves within the lungs, it follows that mere mechanical influences play a much more important part in the pulmonary

circulation than they do in that of the system generally. Hence there can be no obstruction to the blood-flow, either through arteries or veins, within the pulmonary circuit without the blood-pressure within the pulmonary artery being raised. But a rise of the blood-pressure within the pulmonary artery causes the segments of the pulmonary valve to be closed with greater force than usual; the pulmonary second is accentuated. It is probably impossible to detect minor degrees of this accentuation, but as soon as the pulmonary second equals or exceeds the aortic second in intensity of sound there can be no doubt about it.

In the systemic circulation the conditions are very different, it is impossible by any means, experimental or otherwise, to produce such a rise of the general blood-pressure as shall cause an accentuation of the aortic second. In health the aortic second is always louder and more distinct than the pulmonary second, and it is even more difficult to detect any slight increase in its intensity; there is nothing with which it may be compared, or by which the intensity of its sound may be gauged. It is only when some distinct quality is superadded, such as that which is very fairly expressed by the word *booming*, that we can speak with perfect confidence; and my own experience is that whenever an aortic second is heard possessing this quality, some degree of dilatation of the ascending aorta is always present, or if the aorta be not found actually dilated after death it is flabby and dilatable—the *sine qua non* for the production of an accentuated aortic second being the presence in the ascending aorta of a column of blood greater and heavier than usual. *Accentuation of the pulmonary second, unaccompanied by any disease of the lungs, is therefore invariably a sign of some cardiac lesion; and accentuation of the aortic second is as invariably a sign of actual dilatation, or of a flabby dilatable condition of the ascending aorta, when these accentuated sounds are heard in their normal positions,—the third right and the second left costal cartilages.*



Outside of the normal area of cardiac dulness, as depicted on Fig. 3 (p. 24), we occasionally hear the normal cardiac sounds more distinctly than usual, and this may depend either upon increased resonance within a condensed portion of lung lying beneath the spot at which they are heard, or upon the presence of an aneurysmal bulging of the aorta at that spot. In the latter case the normal cardiac sounds may be heard louder than usual at that spot; or if the cardiac sounds be abnormal, these abnormal sounds are more distinctly heard just at that place than they are in its immediate vicinity; or an accentuated second sound alone may be heard in this abnormal position; or this abnormally placed accentuated second sound may be preceded by a localised systolic bruit, or more rarely still the second sound may in this position be replaced by a localised diastolic bruit. *When the cardiac sounds are heard with unusual distinctness outside of the normal area of cardiac dulness, this indicates either disease of the lung or of the aorta, if the sounds are identical with those heard within the cardiac area; but if they vary from these, then they indicate disease of the aorta, probably aneurysm; and in any case they have only to be noted for future more careful examination.*

In the normal condition of the heart the sounds and silences succeed one another in the manner already described, each ventricular systolic sound being accompanied (immediately succeeded, they are not quite synchronous) by an arterial pulse perceptible in the radial arteries. It sometimes happens that these sounds and silences succeed one another with perfect regularity in the cardiac area, and yet a radial pulse may not follow each ventricular systole; the pulse intermits, as it is called. At other times the intermission extends to the cardiac action itself; or the cardiac sounds and silences as well as the radial pulse may all be extremely irregular; and all these phenomena must be carefully observed and noted for subsequent inquiry.

Reduplication of the cardiac sounds is an interesting, and, except in its rarest varieties, by no means an uncommon form

of irregularity. The most extreme and rarest form of this irregularity is when there are four instead of two sounds, two first sounds and two second sounds, the two hearts acting separately, and not simultaneously as usual. More commonly there are three sounds instead of two; two first sounds and one second, which is comparatively rare and seldom to be heard with any distinctness; or two second sounds and one first, which is a matter of common occurrence, and may be heard even in health. Reduplication of the second sound may be heard as a *bruit de rappel* either at the base of the heart, at its apex, or at both. It may be vocalised by the syllables ta-ta, and closely resembles the sound made by a hammer which strikes the anvil, rebounds, and strikes again, remaining motionless. Reduplication of the second sound is occasionally to be heard even in health, under conditions presupposing considerable pulmonary congestion, as after violent exertion, and it is of common occurrence in mitral stenosis in which this congestion is permanent. We know that the increased blood-pressure within the pulmonary circuit is in these cases sufficient to close the valve with a louder snap than usual—to accentuate the pulmonary second; but not infrequently this accentuation is replaced by reduplication. Reduplication of the second sound is by many supposed to be due to *a want of synchronism in the closure of the aortic and the pulmonary valves*; the lightly-filled left ventricle is supposed to empty itself with unusual readiness into the comparatively vacuous aorta, so that the systole of this vessel closes its valve a trifle before that of the pulmonary artery, and thus reduplicates the second sound. As this theory presupposes no alteration in the circulation through the right heart, nor in the closure of the pulmonary valve, it is difficult by it to account for the absence of pulmonary accentuation so conspicuous in cases of reduplicated second; neither does it explain the presence of a reduplicated second when, as occasionally happens, there is only one set of valves to produce it. There is, however, another theory capable of



providing an efficient explanation of reduplication of the second sound in all the various circumstances in which it can arise, and this theory seems the most probable. Ceradini tells us that *the moment the blood-pressure within the artery equalises that within the ventricle the valve segments fall together with an audible snap that initiates the second sound, which is only strengthened, but not produced, by all the succeeding events of the cardiac diastole and arterial systole.*<sup>1</sup> If we adopt this theory and apply it to the explanation of a reduplicated second sound, we see at once how completely it fulfils all the conditions needful to explain the phenomena observed. In the first place, the high blood-pressure within the artery is speedily equalised with that within the ventricle, hence the valve segments snap together in the face of a supporting column of blood, audibly, but without accentuation. In the second place, as the segments of the valve are already in apposition, the arterial systole itself is unable to do more than produce an audible repetition—reduplication—of the second sound, but still without accentuation. In the normal condition of the heart, Ceradini supposes that there is always a snapping together of the valve segments at the close of the cardiac systole, but this is so immediately followed by the arterial systolic closure of the valve that the two sounds are heard as one, the first event being merely strengthened by the second. As this happens both in the aorta and in the pulmonary artery, we may have a reduplication of the second sound in either of those vessels whenever the blood-pressure rises sufficiently to separate the one event from the other. This reduplication may come and go according to the variation in the intra-arterial blood-pressure, the action of both ventricles continuing to be both regular and synchronous. Nay, more, this theory enables us readily to understand how we may have a reduplication of the (pulmonary) second accompanying free aortic regurgitation ; an unusual, but not unknown complication.

<sup>1</sup> *Der Mechanismus der halbmondförmigen Herzklappen* (Leipzig, 1872), S. 62.

Reduplication of the cardiac sounds in disease are more common and more permanent; in health they are more apt to be fugaceous, appearing one instant and disappearing the next. They are abnormal exaggerations of a phenomenon which with care may be detected in every one; reduplication of the first sound occurring at the end of the expiration or commencement of the inspiration; reduplication of the second sound occurring at the end of the inspiration or commencement of the expiration. These normal reduplications depend upon the variation in the pressure produced by the respiratory movements at the origin of the arterial and venous systems.<sup>1</sup> The first sound may be suppressed—in audible—when from any cause the myocardium is enfeebled; this is common in advanced typhus, and not infrequent in weak dilated hearts; it is also said to be a sign of fatty degeneration, but this is certainly not always the case, as many hearts affected in this way have had a distinct first sound to the last (*vide postea sub voce*). The first sound is also frequently in audible when the heart is separated from the chest wall by fluid, as in pericardial effusion. The second sound is less frequently suppressed, but always from a similar cause—some enfeeblement of the cardiac action, or some hindrance to the conduction of the sound to the chest wall.

Within the cardiac area a murmur may take the place of one or other, or even of both of the cardiac sounds, or it may occupy the time of one or other of the silences. A murmur may thus occupy the whole period of a cardiac cycle, or it may occupy any portion of it. Such murmurs may either be of exocardiac or of endocardiac origin, and as endocardiac murmurs are most frequent, and are chiefly due to valvular

<sup>1</sup> Potain, "Note sur le Dedoublements Normaux des Bruits du Cœur," *L'Union Medicale* (1866). In my own experience reduplicated first sounds have been comparatively rare. One of the most perfect instances of this kind was strikingly confirmative of Potain's view, inasmuch as impeded respiration, due to a distended ovarian cyst, was always accompanied by a reduplicated first sound, which invariably disappeared after tapping. This occurred repeatedly. There was no other cardiac abnormality.

incompetence, it is a good rule to endeavour first of all to associate all murmurs with a valvular origin, and only after exhausting this hypothesis to proceed to determine the probabilities in favour of their exocardiac origin, or of their dependence upon some endocardiac cause apart from valvular lesion.

Of all the signs of cardiac disease, murmurs are usually those most implicitly confided in, and yet their evidence must be accepted with caution—first, because we may have murmurs of exocardiac origin which simulate very closely those of valvular origin; and, second, because murmurs truly of valvular origin may disappear temporarily or permanently.<sup>1</sup> Thus we may have a murmur apparently of valvular origin which is really exocardiac; second, we may have a murmur truly of valvular origin, yet without valvular lesion, which may disappear, leaving the heart uninjured; and, lastly, we may have a murmur truly of valvular origin which may disappear temporarily or permanently, the valvular lesion still continuing. It is obvious, therefore, that murmurs cannot of themselves be accepted as certain indications of permanent cardiac lesions, even although we may be able to connect them positively with imperfection of a definite valve, because that imperfection may be only of a temporary and curable character. To this there is but one positive exception, and that is the auricular-systolic, the so-called presystolic murmur, though there is obviously a greater or less probability of any of the other valvular murmurs being also connected with permanent lesion of the valve implicated. That probability, however, falls to be considered when we come to discuss the signs indicative of the lesion of each separate valve. What we have now to consider are the various sounds that may be heard in the cardiac area apart from those we have already learned to recognise as normal, and to point out the measures we adopt to determine the greater or less probability of these sounds being connected

<sup>1</sup> "On the Variation and Vanishing of Cardiac Organic Valvular Murmurs," by W. R. Sanders, M.D., *Edinburgh Medical Journal* (January 1869), p. 584.

with any valvular lesion. Always remembering that no murmur, other than that already referred to, can ever be accepted as a certain proof of actual and permanent cardiac disease, but must be simply noted, to be afterwards duly considered along with the other information derived from inspection, palpation, and percussion, when we come to estimate the probabilities for and against the existence of any special lesion.

When we hear over any part of the area of cardiac dulness any sound which differs from those we have already learned to recognise as the normal sounds of the heart, our first care is to ascertain at which part of the cardiac area this abnormal sound is most distinctly to be heard ; this is termed *the position of maximum intensity*. This position either coincides with one or other of those areas already described as the mitral, the tricuspid, the aortic, or the pulmonary area, or it does not. If the position of maximum intensity of this abnormal sound coincides with one or other of these normal areas, it most probably depends upon some lesion, temporary or permanent, of that valve whose normal sound is audible in that area, and which it either obscures, replaces, precedes, or follows. If this position of maximum intensity does not coincide with one or other of those normal areas, this abnormal sound is certainly not of valvular origin, unless it is heard over the lower part of the sternum, or just to the left of the pulmonary area and in the same plane. And to vindicate its claim to a valvular origin the murmur must be diastolic in the former position, and systolic in the latter ; the reasons for this are to be found in the anatomical relations of the parts connected with the valves at which such murmurs originate, and in the mode in which sound is conducted.

Our next care is to determine *the actual rhythm* of the murmur or abnormal sound, that is *its positive relation to those several physiological acts which constitute a cardiac pulsation or cycle*. To recognise this rhythm we must, first of all, have a



clear conception of those various physiological acts which make up a cardiac cycle; and secondly, we must be able to recognise them. The accompanying figure (Fig. 4, slightly altered from Gairdner<sup>1</sup>) represents diagrammatically the several acts of a cardiac pulsation in the normal adult heart, in which a long sound precedes a short silence, followed by a short sound, and that again by a long silence. So long as

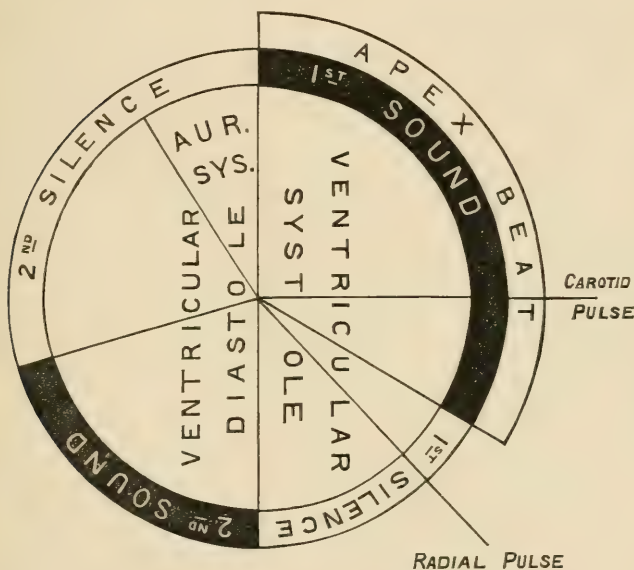


FIG. 4.

the heart-beats are not over ninety per minute, there is never

<sup>1</sup> To Dr. Gairdner we owe the first attempt to represent a cardiac pulsation diagrammatically. Fig. 4 is slightly altered from Gairdner's with the view of making it more physiologically accurate. The apex beat and the two sounds are represented as occupying, as they do, appreciable periods of time during the cardiac cycle. The heart's action is represented as continuous and without actual pause or rest, though at times soundless. The carotid pulse is placed in the position which Valentin says it ought to occupy in relation to the apex beat, and the radial pulse is placed as nearly as possible in its usual position in relation both to the apex beat and the carotid pulse. The foot pulse is nearly synchronous with the radial; according to Mr. A. H. Garrod the difference between the two, with a pulse at 75, amounts to only 0.0012 of a minute.



any difficulty in making out the several relations of these sounds and silences. But when the heart-beats are over ninety per minute, the long pause and the long sound shorten with the increasing rate, so that the sounds and silences gradually lose their distinctively adult character and approximate the uniform tic-tac of the infant, and the more nearly this is the case the greater is the difficulty experienced in determining which sound is long or first, and which is short or second. To ascertain this with precision, we must either wait till rest or treatment has slowed the heart-beats down to ninety, or we must trust to our accuracy in the use of a double stethoscope, knowing that under all circumstances the first sound is always relatively long at the apex and short at the base; or we may determine which sound or murmur is synchronous with the apex beat or the carotid pulse, as that must coincide with the ventricular systole. But we must carefully avoid any reference to the radial pulse as irrelevant and misleading, as even in the normal condition this pulse is separated from the apex beat by an interval which may amount to about one-sixth of a second, and this interval may be increased in disease to a period amounting to the half or even to the whole of an entire cardiac pulsation.

Having determined the position of maximum intensity and the rhythm of a murmur, we are prepared to state the nature of the lesion upon which it depends. For this all we need is simply to remember that, in the normal state of the heart, the auriculo-ventricular valves are closed during the systole of the ventricles, and the arterial valves are open. During the diastole of the ventricles the action of these valves is reversed, the arterial valves are closed and the auriculo-ventricular valves are open. These statements apply to both sides of the heart, which normally act synchronously and simultaneously. When we hear a murmur with a position of maximum intensity in the mitral area, we know it to depend upon some defect of the mitral valve. If this murmur be synchronous with the systole of the ventricles, it

depends upon some defect in the closure of that valve, and is a murmur indicative of regurgitation of blood backwards into the auricle. But if this murmur be synchronous with the diastole of the ventricles, it depends upon some obstruction to the flow of the blood through the open mitral valve, and is, therefore, a murmur of obstruction. *A murmur of mitral regurgitation runs off from the apex beat, and more or less completely replaces the normal first sound. A murmur of mitral obstruction accompanies or follows the second sound, but in no respect interferes with its production.* This murmur of obstruction to the onward flow of the blood at the mitral valve may be audible throughout the whole of the ventricular diastole; more usually it is only to be heard at an appreciable interval after the second sound, running up to and ending with the first sound. From this circumstance this murmur is very generally termed a presystolic murmur. A reference to the diagram (Fig. 4) shows that, as physiology teaches, the period of time immediately preceding the commencement of the ventricular systole is occupied by the systole of the auricles, so that a so-called presystolic murmur is really auricular-systolic in rhythm. It is not only a murmur of obstruction to the onward current of the blood, but it is also a murmur of obstruction to direct cardiac action, and, like all such murmurs, it is always rough in character. It is the only generally recognised cardiac murmur which is invariably associated with actual disease of the valve affected.

These three murmurs—the mitral systolic, the mitral diastolic, and the presystolic or auricular-systolic—may each be heard separately in separate cases; or any two of them may be present; or more rarely all three are to be heard together, and then we have a murmur whose position of maximum intensity is in the mitral area, and which runs right through the cardiac cycle. In all these instances the first sound is either much altered or entirely replaced by the murmur; while the second, though not always to be heard at

the apex, is always to be heard at the base of the heart, and the pulmonary second is always accentuated and sometimes very greatly so, unless, indeed, we hear two seconds immediately following each other forming the reduplicated second just described, when accentuation of the pulmonary second is always much modified, and often entirely absent. These murmurs are most common on the left side, but may also be heard over the right heart. A systolic tricuspid murmur is of frequent occurrence, a presystolic tricuspid is occasionally, though rarely, to be heard, but a diastolic tricuspid murmur, though possible, is yet, so far as I am aware, hitherto unrecorded. In making this statement I refer to a diastolic murmur clearly attributable to the right auricle or ventricle alone, both pulmonary and aortic second sounds remaining distinctly audible, and with entire absence of any symptom or sign of aortic regurgitation. This is a very necessary warning, because it often happens that a diastolic murmur, arising from defective closure of the aortic valves, is heard loudest over the lower part of the sternum, partly conducted downwards by the descending current of the blood, and partly by the resonating properties of the sternum. We shall afterwards see that a diastolic murmur, of purely aortic origin, may have its position of maximum intensity over the lower part of the sternum—in the tricuspid area—or even in the mitral area itself, and that this is said to depend upon the segment of the valve affected.

At the base of the heart the murmurs audible seem to be less complicated than at the apex, because at the base there is no presystolic murmur, and the only possible murmurs are either systolic or diastolic. But the causes of these are so various as to give rise to much speculation, and the pulmonary region has not undeservedly been called the region of romance, from the variety of theories propounded in explanation of the murmurs to be heard there. While the physical causes of aortic murmurs, and the physical relations of the aorta itself are so complicated, and so

efficiently modify the conduction of these murmurs, that though it is quite usual to say that a systolic murmur, having its position of maximum intensity at the sternal end of the second right costal cartilage, is due to aortic obstruction, this is by no means always the case. And in like manner, though a diastolic murmur, audible in the same position, is most probably due to aortic regurgitation, yet the absence of such a murmur cannot be accepted as proof of the absence of regurgitation, because the murmur due to regurgitation is sometimes only audible beneath the level of the aortic valves.

In noting the characteristics of any murmur, the chief points to be determined are, first, *its position of maximum intensity* as a probable indication of the valve affected; second, *its rhythm*, presystolic, systolic, or diastolic, indicating its position in the cardiac cycle; and lastly, we must note what has happened to the *valve sound normally heard* in any given position, whether it is *wholly replaced by the murmur*, or is *only altered in duration and character*. We must also remember that even the presence of a well-defined murmur is not by any means a certain indication of actual lesion of the valve implicated, it may only signify incompetence without either disease or deformity. Neither are we justified in regarding the absence of a murmur as any certain proof of the non-existence of valvular disease. The characteristics of a murmur, and its relations to the normal sounds and silences, must merely be noted and afterwards duly considered in connection with the other physical signs already commented on.

Endocardiac murmurs of other than valvular origin are occasionally, though rarely, observed. Sometimes these murmurs are due to congenital deficiency of some part of the septum between the two hearts; and in that case they only intensify those valvular murmurs which almost in variably coexist, and extend the area of their audibility. Where no valvular lesion exists, as in those rare and



problematical murmurs said to occur, in a heart otherwise healthy and normal, from the presence of a flake of lymph or other vibrating substance attached to one of the *cordæ tendineæ*, the position of maximum intensity and the rhythm may closely simulate those of a valvular lesion. But a due consideration of the other physical signs associated with them, as well as of the physical facts already pointed out as indicative of actual or constructive obstruction to the onward flow of the blood, will be sufficient to enable us to avoid the commission of any glaring error in diagnosis.

Exocardiac murmurs frequently simulate in their timbre those of endocardiac origin, and when they also coincide in rhythm with the systole or diastole of the heart, there may be some little difficulty in distinguishing such a murmur from one of endocardiac origin. Exocardiac murmurs, however, are chiefly due to friction of the roughened visceral and parietal portions of the pericardium upon each other, or of the pericardium upon the pleura, and, unless in very exceptional circumstances, this friction is always greatest over the centre of the heart, and neither at its apex nor exactly at its base. An exocardiac murmur, therefore, does not coincide in position of maximum intensity with any of the recognised valvular areas. It has a distinctly superficial character, it radiates equally all round its place of origin, but never to any great distance. It is not propagated along any blood-current, nor is it associated with any accentuation of the second sound, nor with any of the physiological indications of obstructed circulation. Moreover, a friction sound only rarely simulates the soft blowing murmur of valvular incompetence, more often it resembles the crackling of parchment, the creaking of new leather, or a mere grating or rubbing sound of greater or less intensity. Whatever its timbre or rhythm, we must treat it as we ought to treat every sound heard over the cardiac area, by carefully noting its character, position of maximum intensity, rhythm, and direction in which it is propagated, to be afterwards sub-



mitted, along with any other facts ascertained, to a careful and discriminating scrutiny.

A similar remark may be made in regard to those murmurs audible in various positions out of the cardiac area, and which owe their origin to the passage of the blood through the vessels. Such murmurs are sometimes heard in the arteries and at others in the veins. They are chiefly systolic in rhythm, but at others they are diastolic, or they may continue throughout the whole cardiac cycle. In the arteries these murmurs may be strictly localised, or they may be audible over every part of the arterial system. In the veins, as a rule, they are audible everywhere, provided certain conditions are complied with, though they are only permanently present in certain parts of the body where these conditions exist naturally. Such intravascular murmurs are of the most various significance, and can only be most briefly referred to now. Sometimes they signify serious lesion of the vessels themselves; at other times, nothing worse than some slight deformity of the chest, probably the result of rickets; and at still other times, only an alteration in the constitution of the blood itself, which may arise from various causes.

I cannot conclude this lecture without a cursory reference to the manner in which these murmurs are believed to be produced, not only because this is a subject interesting in itself, but also because distinct and accurate ideas as to the causation of murmurs must necessarily lead to greater accuracy in diagnosis. The crude notions of our forefathers culminated in the opinion that a murmur is produced by friction of the blood-current on its containing walls. This idea has been completely set aside by the researches of Poiseulle, and others, which show that however rough the bed of the blood-current may be it flows smoothly over it, without giving rise to any friction whatever. Corrigan was the first to start the theory that murmurs occurring in the course of the circulation are due to vibrations produced by

eddies in the blood-column itself. This theory has been reduced to scientific simplicity by Mr. A. Chaveau,<sup>1</sup> who has with great probability referred the causation of all murmurs, whether intracardiac or intravascular, to the

<sup>1</sup> *Comptes Rendus de l'Académie des Sciences* (1858).—"Toute veine fluide est le siège de vibrations susceptibles de produire des sons, vibrations qui ébranlent aussi l'orifice d'écoulement de la veine"—p. 841. Even the intravascular murmurs of anæmia (spanæmia) are due "aux vibrations de la veine fluide intravasculaire, vibrations qui se produisent quand le sang pénètre avec une force suffisante dans une partie réellement ou comparativement dilatée de l'appareil circulatoire"—p. 933.

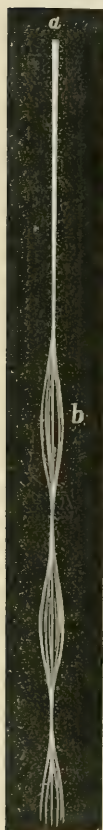


FIG. 5.

The nature of a fluid vein may be more readily understood from a short account of the conditions under which it is known to be formed. When fluid escapes through a perforation in a thin plate forming the bottom of an open vessel kept constantly full, Torricelli supposed that the efflux could be ascertained by the following theorem:  $Q = ts\sqrt{2gh}$ , in which  $Q$  represents the outflow,  $t$  the time occupied,  $s$  the sectional area of the orifice,  $g$  the acceleration of gravity, and  $h$  the height of the surface above the orifice. The actual outflow, however, is, when measured, always about one-third less than that given by calculation, and the discrepancy has been found to be due to the fact that the velocity, on which the calculation is based, is only true of the central part of the jet, the outer part of this being retarded by the interference of the currents converging from all sides of the vessel. The convergence of these currents also causes the jet to taper off to  $\frac{1}{6}$  of its original dimensions at a distance from the orifice of one-half its diameter, and if the sectional area of this narrow portion, the *vena contracta*, as it is termed, is substituted for that of the original orifice, the actual outflow is then correctly expressed by Torricelli's theorem. Up to this narrowed portion the jet is clear and pellucid, but at this point, probably from the conflict between the tendency to diverge inherent in the outer currents and the attractions of cohesion and gravity, the jet loses its transparency and breaks up into those vibrating fluid veins the phenomena of which have been described by Felix Savart as follows. The part of the vein *ab* (Fig. 5) is steady and limpid, presenting the appearance of a solid rod, which obstructs vision and wets the finger when

it is passed through it. This limpid rod decreases in diameter till it reaches a point of maximum contraction (just above *b*), beyond this it is turbid and unsteady, marked by periodic swellings and contractions, does not wet the finger when passed through it, and does not obstruct vision, even though the fluid be mercury. At *b*, in fact, the liquid is no longer continuous, but resolves itself into a series of liquid spherules, which have an appearance of turbid continuity, from their rapid succession not permitting the primary impression to fade from the retina till succeeded by a second. Fig. 6 shows this fluid vein illuminated by an electric flash, the drops of which it is com-

sonorous vibration of fluid veins, such as have been observed by Savart, and which are capable of being transferred, as Marey has experimentally shown, to the surrounding fluid within which the vein has formed, as well as to the vessel containing it. The roughness of the murmur and the tactile perceptibility of the vibrations are in some degree proportionate to the force exerted in producing this sonorous vein, while its musical character must depend upon the rapidity of these vibrations, as well as on the physical structure of the parts to which these vibrations are communicated.

But the mode in which the production and conduction of these sonorous vibrations may be modified by evident or imperceptible alterations in the action of the heart itself, or by changes in the anatomical relations of the parts concerned, produced by alterations in the posture of the patient himself, are problems yet to be investigated. It may be that a careful and experimental examination of these problems may be of the utmost value in promoting a more accurate diagnosis of cardiac and vascular diseases.

posed are seen as it were motionless in the air, and the cause of the periodic swellings and contractions is at once apparent. For the drops when first detached are seen to have their long axis vertical, abandoned to their own molecular forces they seek to become spheres, and, like a pendulum in motion seeking to return to rest, their contraction goes too far and they become flattened spheroids, which again elongate vertically. Hence the appearance of alternate swelling and contraction. Savart traced the production of these pulsations to the orifice through which the fluid vein passed, but did not regard them as the result of friction. Under moderate pressure they succeed each other sufficiently rapidly to produce a feeble musical note, the pitch of which may be determined by allowing the drops to fall upon a stretched membrane. *Vide* Tyndall *On Sound* (London, 1869), p. 224, etc. By attaching tubes of various forms to the original orifice, the formation of a fluid vein may be completely prevented, and the outflow may even be increased beyond the quantity calculated by Torricelli. It seems not improbable that the relations of the several parts of the circulatory system to each other may be so adjusted that sonorous fluid veins are only formed when disease of the vessels, or some change in the constitution of the blood, has altered these normal relations.



FIG. 6.

## LECTURE II

### ON INCOMPETENCE OF THE AORTIC VALVE, WITH SPECIAL REFERENCE TO THE DIAGNOSTIC SIGNIFICATION OF THE MURMURS

THERE is no disease of the heart, of common occurrence, which possesses so many elements of interest as aortic incompetency, whether we regard its pathology in relation to the many causes which may give rise to it, to the concomitant morbid alterations in the aorta, in the heart itself, and in the circulation, which may be so various; or whether we regard its diagnosis, which involves a careful discrimination of the causes of the incompetence, as well as of the consecutive changes in the heart, if we wish it to be of value in its application to either prognosis or treatment.

In aortic incompetence, as in all cardiac affections, our information is derived from the state of the pulse, and from the inspection, palpation, percussion, and auscultation of the patient. A due consideration of the information thus derived enables us to predict with considerable certainty the condition of the aortic valves, and to determine the degree in which the walls of the heart and the circulation generally have been affected by the persistence of this abnormal condition.

Confining our attention for the present to the phenomena revealed on auscultation, you are aware that the murmur indicative of aortic incompetence is a diastolic murmur of a more or less soft and blowing character, audible, as is usually said, at the base of the heart. But this is by no means



always the case. Sometimes this diastolic murmur is only audible, and that but faintly, just below the aortic valves, at mid-sternum, a little beneath the level of the third rib. Very rarely, it is loudest at the left apex;<sup>1</sup> very frequently, it is loudest and sometimes only to be heard—except in the arteries—at the ensiform cartilage, or more nearly in the position of the right apex. At times it is loud and distinctly audible over every part of the cardiac area, and then it is also to be heard in every part of the arterial system to which the stethoscope has access. While at still other times it is inaudible above the level of the aortic valves, and is not to be heard in any artery. I have already mentioned that a murmur is produced by the formation of a fluid vein at any point in the blood-current,<sup>2</sup> and a moment's reflection shows us that, in most cases, the same cause that produces a fluid vein at the aortic orifice during the diastole of the heart, must be equally potent during its systole. Hence in most cases of aortic incompetence we have a double murmur, a *bruit de va et vien*, a see-saw murmur of which the first or systolic portion is usually rough, and the second or diastolic portion always soft and blowing, though sometimes the one and sometimes the other may be loudest. The propagation of the systolic portion of this double murmur is subject to the same vagaries as that of the diastolic portion, being at one time audible all over the cardiac area, at another time only to be heard at and above the aortic orifice, and yet again it is chiefly audible in the arteries alone. The term double aortic lesion is thus a common and, as we see, by no means an inapplicable designation for that affection of which incompetence of the aortic valves forms always the most distinctive though rarely the sole important feature. The variations in

<sup>1</sup> This position of maximum intensity of the murmur of aortic incompetence is said to be an indication of rupture or disease specially affecting the posterior or mitral segment of the semilunar valve; *vide* Dr. Balthazar Foster's *Essays on Clinical Medicine* (London, Churchill, 1874), p. 121. Most observers are, however, agreed that this idea is highly problematical.

<sup>2</sup> *Vide antea*, p. 46.



the manner in which the constituents of this double murmur are propagated are not mere vagaries in the conduction of sound, they are important facts depending upon physical causes, and convey most valuable information to the understanding mind of him who hears.

The following cases have been selected to show the chief varieties of aortic regurgitation, and the manner in which the condition of the valves influences the production and propagation of the murmurs:—

CASE I. Janet M'Gowan,<sup>1</sup> aged twenty-two, admitted 25th March 1874, to Bed 2, Ward XIII., complaining of subacute wandering pains in her joints. This attack commenced twelve months ago, and was at first accompanied by great pain and some swelling in the region of the heart, for which a fly blister was prescribed; after this the swelling disappeared, and the pain subsided, but it has never altogether ceased. Three years and a half ago she had an attack of rheumatic fever, but apart from that has always enjoyed good health, except suffering from measles and scarlet fever when a child. The patient is well developed; expression natural; integuments moist; temperature 98·6°; limbs somewhat wasted; joints noways deformed, but occasionally painful, chiefly the shoulder and elbow joints; no anasarca; arteries and veins normal on inspection; pulse 73, slightly jerking, but this is not perceptibly increased on elevating the arm at right angles to the body as the patient lies in bed. On percussion the cardiac dulness, at one inch from the left edge of the sternum, extends vertically from the upper border of the third rib till it meets the tympanitic resonance of the stomach, and extends, in the line of the fourth rib, from three-quarters of an inch to the right of the sternum, transversely across the chest, for a distance of four inches and three-quarters. The heart's apex beats firm and full between the fifth and sixth ribs, two inches and three-quarters to the left of the sternum. On auscultation in the mitral area the first sound is heard

<sup>1</sup> Condensed from the notes of Mr. J. H. Clarke, clinical clerk.

loud, thumping, and accompanied by a systolic murmur ; no diastolic sound nor murmur is audible. In the tricuspid area the first sound is loud, distinct, still somewhat thumping, and accompanied by a systolic murmur, followed by an equally distinct diastolic one. Above the fourth rib the thumping character of the first sound is lost, while the systolic murmur increases in intensity. In the aortic area neither a systolic nor a diastolic sound is audible, they are replaced by a very loud and distinct double murmur ; the systolic portion of this murmur is extremely rough, and propagated with great distinctness into the carotid arteries, while the diastolic portion is quite inaudible in them. In the pulmonary area a double murmur is heard, the systolic portion of it entirely replaces the first sound, while the diastolic portion partially obscures an accentuated second sound. Her respirations are 32 per minute ; no cough, and no dyspnœa, except on exertion, and then not to any great amount ; pulmonary physical signs normal. Nervous system normal. Appetite defective ; bowels regular ; urine normal, specific gravity 1015 ; menstruation regular till twelve months ago, since then irregular. On 15th May it is noted that a well-marked presystolic murmur has been developed, and this continued till the patient was sent to the Convalescent Hospital on 20th May, completely relieved of her rheumatic symptoms.

You will observe that this patient came under treatment solely for her subacute rheumatic affection, and though labouring under serious cardiac disease, which will undoubtedly shorten her days, this had been so completely compensated as to be altogether mute. The hypertrophy of the left ventricle was sufficient to counterbalance its dilatation, and also to compensate the obstruction to the circulation due to the regurgitation through the incompetent aortic valves. For the present I omit as irrelevant all reference to the state of the mitral valve, and shall direct your attention solely to the aortic murmurs present, and to their mode of propagation.

You will note first of all that the systolic murmur, more

or less audible over the whole cardiac area, has its position of maximum intensity in the aortic area, and is therefore clearly of aortic origin. But the essential part in the production of a murmur is the formation of a fluid vein at the point of origin—here the aortic orifice, and to produce this fluid vein there must be some real or constructive constriction at this point. Either the aortic orifice must be obstructed, or the aorta beyond must be dilated so as to produce a relative constriction. In this case there is no increase of dulness over the upper part of the sternum, and no pulsation in the tracheal fossa; no sign, therefore, of any actual dilatation of the aorta. Is there any reason to suspect any relative dilatation due to actual obstruction at the aortic orifice? The answer to this is not difficult. From the history of this patient there is every reason to believe that she has had an attack of inflammation of the pericardium, this we deduce from the swelling and pain in the præcordial region present during the early period of her rheumatic attack. The more or less constant cardiac pain accompanied by a thumping first sound, and the subsequent development of a presystolic murmur, conclusively prove that this pericarditis has been associated with a more or less chronic endocarditis that has stiffened and deformed the mitral valve. It is natural to suppose that a similar effect has been produced upon the aortic semilunar valves. The result of the thickening and deformity of these valves is that they obstruct the onward current of the blood, causing an actual constriction of the aortic orifice and a relative dilatation of the aorta beyond, just the condition needful to give rise to a fluid vein when the blood is forced through this constricted orifice by the ventricular systole. This fluid vein gives rise to an audible murmur, systolic in time, and rough in proportion to the disproportion between the size of the blood-wave and the orifice through which it is thrown, as well as to the ventricular force exerted in expelling it. In accordance with the laws of sound, a murmur thus produced is carried with most dis-

tinctness with the onward current of the blood, and is to be clearly heard in the carotid arteries; it is also propagated with greater or less distinctness, according to circumstances, in every other direction. The history of this case is thus in complete accord with this most probable explanation of the systolic portion of the aortic murmur, and we may accept it without hesitation.

But if the aortic valves are so thickened and crumpled as to obstruct the onward flow of the blood, they can scarcely be expected to be perfectly competent. They may be competent, but this is unusual. Most generally, as we can readily suppose, such crumpled valves do not meet accurately, they are incompetent, and then the same cause that gave rise to a fluid vein and a murmur during the systolic outflow, gives also rise to a fluid vein and a murmur during the diastolic regurgitation of the blood into the ventricle. The diastolic murmur is always much softer and more blowing than the systolic aortic murmur, because the forces that conduce to regurgitation are much less powerful than the systole of the left ventricle. Evidently this has been the course of events in this case. We are not yet in a position to explain, in every case, why a diastolic murmur originating at the orifice of the aorta should at one time be heard loudest above that part, as in the so-called aortic area, and at another time lower down the sternum. At present we can only state it as a fact, and indicate the probability that the reason will ultimately be found to be closely linked with the nature of the obstruction, as well as with the readiness with which the vibrations of the fluid vein are propagated outwards through the wall of the vessel in one case, and downwards with the regurgitating blood in another. There seems some reason to suppose that the greater the obstruction and the more vibratile its connection with the arterial walls, as well as the firmer it is in texture, the more likely the murmur is to be localised in the aortic area. When the obstruction is less, and less vibratile in its structure and



connections, the murmur tends rather to be propagated downwards with the blood-current, or possibly it may first develop as sound at that part of the heart on which the fluid vein impinges, propagation in other directions ensuing in accordance with the laws of sound. These attempts to explain the reasons for the different directions in which aortic murmurs are propagated have, at least, probability on their side, and the more we inquire into the various forms of aortic incompetence the greater will this probability appear. In the present case both the systolic and diastolic murmurs are most distinctly to be heard in the aortic area. The incompetence of the aortic valve is not very great—(1) Because the apex beats in its normal plane, though a little outside of its normal position; the heart has more of the purse shape usual in mitral disease, than of the conoid form, due to dilatation of the left ventricle, so common in aortic incompetence. (2) Because there is but little jerking to be felt in the pulse or seen in the carotid or brachial arteries. This jerking of the pulse is due to the marked contrast between the rapid distention of the arteries with a large systolic blood-wave, and the sudden collapse that accompanies cardiac diastole; it signifies free regurgitation and a strong and capacious left ventricle. The absence of jerking is not in this case, as in some, due to mere weakness of the ventricular wall, because the apex of this heart beats both firmly and forcibly, but it is evidently due to the absence of any great dilatation of the left ventricle, and, as an ultimate cause, to the absence of any great aortic incompetence. And lastly, the aortic incompetence is not great because, though the diastolic murmur is so loud at the base of the heart, it is quite inaudible either in the carotid or crural arteries. We shall presently see that a diastolic murmur may be audible in the arteries even though inaudible at the base of the heart, and that a diastolic murmur so propagated is invariably associated with all the signs and symptoms of great valvular incompetence; in the present case the diastolic murmur is loudest in the



aortic area, and inaudible in the arteries, and the signs and symptoms all indicate a trifling degree of incompetence. There are therefore strong reasons for believing that in this case—confining our attention to the aortic valves—there is a stiffened and somewhat shrivelled or crumpled condition of these valves due to rheumatic valvulitis. This is the fundamental lesion. The valves may be also atheromatous, or covered with dense vegetations, possibly both, but of these possible conditions we have no indications; on the other hand, the incompetence due to their shrivelled condition is not great, but the coexistence of the mitral lesion makes the prognosis more grave than it would otherwise have been.

The following somewhat similar case is an example of the manner in which murmurs of aortic incompetence vary even in cases which are fundamentally alike, it shows also that these variations are probably due to slight alterations in the physical causes of these murmurs, which only require a larger and more accurate experience to enable us to interpret correctly :—

CASE II. Mary Anderson,<sup>1</sup> married, a laundress, aged thirty-one, admitted to Ward XIII., 3rd December 1872, complaining of severe pain in the region of the heart, a choking sensation in the throat, a troublesome cough and spit, and complete loss of sleep. Nine years ago she suffered from a sharp attack of rheumatic fever lasting eight weeks, six of which were passed in hospital. At that time she had severe pain in the region of the heart, both in front and behind, accompanied by palpitation. A fortnight after dismissal she was able to return to work. A similar but less severe attack has recurred each winter since that time. The present attack commenced eight weeks ago with severe pain in the cardiac region, swelling of the legs, and a choking sensation in the throat, which did not interfere with swallowing. She also suffers from atonic dyspepsia, temporarily relieved by vomiting; and the amount of urine passed has gradually diminished.

<sup>1</sup> From the notes of Mr. O'Connor, clinical clerk.

Patient's family history is good. She has two children, both alive and healthy. Her face is somewhat livid, her fingers slightly clubbed, her legs œdematous, the joints normal, temperature 99°, sensation and intelligence normal. She has slept well the two nights following her admission. Her pulse is 112, irregular, somewhat forcible, and felt to be distinctly jerking on elevating the arm at right angles to the body as she lies in bed. Considerable pulsation is visible at the root of the neck, both in the carotids and in the tracheal fossa. No venous pulsation is to be seen. The apex beats somewhat forcibly between the sixth and seventh ribs, about three and a half inches from the left edge of the sternum. In the parasternal line, one inch from the left edge of the sternum, dulness begins at the upper border of the second rib and extends down to the liver dulness. Transverse dulness in the line of the fourth rib commences at three-quarters of an inch to the right of the sternum, and extends across for four inches and three-quarters. On listening over the apex beat, a slight thump is heard, forming, as it were, the last portion of a **presystolic murmur**; the first sound is obscured by a murmur. The second sound in this area is replaced by a soft diastolic murmur. In the tricuspid area the first sound is distinctly audible, notwithstanding that the obscuring murmur is even louder there than in the mitral area; the diastolic murmur is still soft, but also more distinct than in the mitral area. In the aortic area, the first sound, audible in both the mitral and tricuspid areas, is completely replaced by a systolic murmur which is loud and rough, and attains its maximum intensity in this position; the second sound is wholly wanting, being replaced by a soft, low, but distinct diastolic murmur, which has its position of maximum intensity at mid-sternum, just below the fourth rib. In the pulmonary area, the first sound is wholly replaced by a loud systolic murmur, and the second sound is heard loudly accentuated, but somewhat obscured by the diastolic murmur propagated across from the aortic area. In the carotid and femoral arteries a loud systolic murmur

alone is audible; the diastolic murmur is quite inaudible. The respirations are 32 per minute. The cough is sharp and harsh, accompanied by a slight watery expectoration. The pulmonary percussion is everywhere normal. On auscultation crepitation is heard over both lungs in front; behind this is mingled with occasional rhonchi. The tongue is clean, but red and raw-looking at the tip. There is great craving for food, some flatulent distention of the stomach, especially after a meal; bowels irregular; liver dulness normal. Patient has not menstruated for twelve months. Her urine varies in quantity from 36 to 52 oz. of a clear straw colour; on admission there was a trace of albumin, but this has now disappeared.

The history of this patient is that of rheumatic fever, accompanied by an affection of the heart, which inspection, palpation, and percussion agree in showing has resulted in dilatation, particularly of the right auricle and of the left ventricle, with some hypertrophy of the latter. The slight thump preceding the first sound is readily recognised as the last remaining portion of a presystolic murmur, quite as distinctive to an educated ear as a well-marked auricular-systolic murmur, and equally with it a sign upon which it is safe to rely as proof of existing stenosis of the mitral opening. Mitral stenosis is a common result of rheumatic endocarditis, and of itself a sufficient cause of the pulmonary congestion and œdema, as revealed by the accentuated pulmonary second, the cough, and the watery expectoration, —as well as of the great interference with the systemic circulation evinced by the livid countenance, the œdema of the lower extremities, the albuminuria, and the flatulent dyspepsia. Stenosis of the mitral opening has, however, no influence in promoting dilatation of the left ventricle, rather the reverse, so we must look elsewhere for the cause of this. It is not difficult to find. In the aortic area there is a loud systolic murmur, with its maximum intensity in that position. This is propagated with great distinctness into the arteries, affording proof that the same process that has deformed the

mitral valve has also thickened and stiffened the segments of the aortic valves, and made them a hindrance to the egress of the blood from the ventricle. In the same area we have also a soft diastolic murmur replacing the aortic second, showing that the valvulitis which has thickened the valves has also shortened them and made them unable to close the orifice, has made them incompetent. This incompetence, though sufficient to give rise to a murmur audible at and below the aortic orifice, is yet so trifling and has so little influence on the arterial circulation that the diastolic murmur is not to be heard in the carotid arteries. It seems to be conveyed to the aortic area chiefly through the bones (sternum and ribs), but it is more readily conveyed downwards, because in this direction the regurgitating blood-current aids the conduction of the sternum. An incompetence so trifling would be readily compensated in an otherwise healthy heart, in the present case it is uncompensated, and greatly intensifies the evil influence of the mitral stenosis. Together they form an obstruction to the circulation sufficient to account for all the serious symptoms that are present, as well as to make the prognosis most grave in this patient already enfeebled by repeated rheumatic attacks.

The patient was treated with nourishing unstimulating diet, with half-ounce doses of the infusion of digitalis and 5 grains of carbonate of ammonia thrice a day, 15 grains of chloral being given at bed-time to allay her cough and cardiac pain, and to procure sleep. At first she did well, but towards the end of December (21st) she began to complain of pain in her joints, the oedema of her legs increased, and there was also considerable pain over her stomach and liver, the latter organ commencing to enlarge and to become tender to touch.

She now received 10 minims of the tincture of digitalis, with 5 minims of the liquor arsenicalis and a similar quantity of the tincture of the perchloride of iron, three times a day. She gradually got worse; in the beginning of



January diarrhœa set in, but was kept in check by appropriate treatment. The pain in the chest, the breathlessness, and the pain over the liver continued, and she died at 1 A.M. on the 15th of January 1873.

*Autopsy.*<sup>1</sup>—17th January. Heart considerably enlarged; mitral opening constricted, admitting only one finger; aortic valves atheromatous, the cusps covered with vegetations. Several hæmorrhagic infarctions in the lungs, which are otherwise healthy. Liver weighs 4 lbs. 14 oz., is of firm consistence, slightly hypertrophied, and exhibits all the characteristics of extreme chronic congestion. Spleen weighs 11 oz., firm and congested. Kidneys are slightly congested, and exhibit several depressions, the result of previous infarctions; the right kidney weighs 6½ oz., and the left 5½ oz. Other results unimportant.

These two cases may be accepted as examples of the character of the murmurs, and the manner of their propagation, in those whose aortic valves are crumpled, thickened, and atheromatous, as the result of rheumatism in the young, or of more chronic disease in those who are older. Such valves are too stiff to be moved out of the way by the blood-current, consequently they narrow the opening and give rise to the formation of fluid veins which are the cause of a loud systolic murmur, carried by the blood-current into the arteries, always therefore audible in the carotids without any compression of their calibre, and dying gradually off within the arteries as the distance from the heart increases. These thickened valves are not much moved by the systolic cardiac current, they have no distance to fall, and the cusps being no longer membranous, every source of tensile vibration is wanting. There is no second sound, not because the valves are destroyed,—they still exist, larger and thicker than in health, though imperfect. Under other circumstances we may have a second sound accompanied and followed by a

<sup>1</sup> From the *Pathological Records of the Royal Infirmary*. Weight of heart not recorded.



diastolic murmur; in such cases as the present we have no second sound because the cusps are immobile and have entirely lost their membranous elasticity. The deformity of the cusps, however, prevents the valve from closing perfectly, hence there is regurgitation, the formation of fluid veins, and a diastolic murmur entirely replacing the second sound. As the valves still obstruct the regurgitation it is seldom great; the diastolic murmur, however loud it may be, is never propagated into the carotid arteries. It may be heard all over the cardiac area, chiefly propagated through the bones, the sternum acting as a sounding board. Its position of maximum intensity is usually about mid-sternum, just below the fourth rib, and under all circumstances it is propagated downwards with most distinctness. In the class of cases described the essential characteristics of the physical signs *quoad* the murmurs are, *the loudness of the systolic murmur in the aortic area and its ready propagation into the carotids; while the diastolic murmur is most distinctly heard just over the aortic valves, or immediately below them*, and it is never propagated into the carotid arteries, or only in the faintest possible manner. Propagation of the diastolic murmur into the arteries is always to be regarded as an indication of considerable regurgitation.

The next case I shall relate presents phenomena which are exactly the reverse of those just described, and indicate a lesion of a totally different character.

CASE III. James Bailey,<sup>1</sup> aged thirty-five, unmarried, a labourer residing in Leith, was admitted to Bed 2, Ward V., on 19th February 1874, complaining of a severe cold, constant cough, and pain in the epigastrium. The patient says that he has been short of breath as long as he can remember, and he thinks this has got worse as he grew older. Twenty years ago he had fever (probably typhus), for which he was treated in this infirmary. Nine years ago he was for one month under treatment in this infirmary for rheumatic fever; during

<sup>1</sup> From the notes of Mr. A. Field, clinical clerk.

this illness he was delirious for two or three nights. About three years ago he was again in this infirmary for a less severe attack of rheumatism. He has always been well fed, comfortably housed and clothed, but his occupation necessitates constant exposure to all kinds of weather. About three weeks ago he was more than usually exposed to severe weather, and except the dyspnœa, all his ailments date from this time. Patient is fairly well developed, height 5 feet, weight 8 stone 9 lbs., muscularity good. He usually sleeps on his left side, but is often obliged to sit up in bed for a time on account of his dyspnœa. Expression of his face is normal, integument normal, temperature 98°, limbs and joints natural; pulse 75 per minute, full, jerking, and delayed about half a pulsation (cardiac cycle) behind the apex beat; very evident pulsation is to be seen in both carotids and in both brachials. On inspection, the apex is seen to beat with tolerable distinctness in the seventh intercostal space, five inches to the left of the xyphoid cartilage. From this an undulatory pulsation is diffused up to the fourth rib, the most remarkable part of which is a systolic depression, greatest between the fifth and sixth ribs. In the *scrobiculus cordis* a slight pulsation is distinctly visible, and this is seen to be communicated to the entire hepatic region. On palpation, the diffuse apex beat is felt to extend over several inches; this diffuse apex, as well as the whole left ventricle, communicates a heaving, but not forcible impulse to the hand. In the parasternal line, dulness commences at the third interspace and extends downwards to the liver dulness. At the level of the fourth rib, the transverse dulness extends from half an inch to the right of the sternum to half an inch beyond the left nipple, a distance of six inches and a half. On auscultation in the mitral area, the first sound is obscurely heard, and of an impure quality, but without distinct murmur; the second sound is heard obscured by a diastolic murmur. In the tricuspid area, the first sound is much more clear and distinct than in the mitral area; no second sound is audible,

it is completely replaced by a loud and well-marked but soft diastolic murmur. In the aortic area the first sound is obscurely heard, much as at the apex, and without distinct murmur; the second sound is replaced by a distinct diastolic murmur. In the pulmonary area, the first sound is impure; the second sound accentuated, but obscured by the diastolic murmur. The diastolic murmur is heard over every part of the chest, but is loudest over the sternum, beneath the aortic valves; it is even to be heard over the chest posteriorly. In the carotid and femoral arteries, a well-marked and distinct double murmur is readily heard. The percussion of the lungs is everywhere normal, but on auscultation a good many rhonchi and crepitant rattles are heard distributed over them. Liver dulness is normal; and except some flatulent, atonic dyspepsia, there is nothing else of importance about this patient. Bailey continued gradually to improve under treatment till 14th April, when he was discharged. Since then he has written from Ireland to say that he has given up all remedies, and feels quite well. The treatment consisted mainly in the administration of digitalis, at first in the form of half-ounce doses of the infusion, with 5 grains of the carbonate of ammonia, three times a day. Latterly the tincture was given with iron and arsenic. The lung symptoms were the result of a severe catarrh engrafted upon a state of chronic pulmonary congestion, due to the aortic insufficiency, and were only of consequence as an aggravation of his serious heart affection. There were several very interesting points in this case: (1) The great dilatation and hypertrophy of the left ventricle, revealed by the low position of the true apex, which beat beneath the edge of the eighth rib, the general slow heaving impulse of the ventricle, and the systolic dimpling of the surface of the ventricle, evidently due to displacement of the lung by the large ventricle, and the pressure of the atmosphere on the walls of the chest during the systolic recession of this large ventricle. (2) The absence of any special implication of the right ventricle,

notwithstanding the great amount of disease upon the left side. (3) The impurity of the first sound in the mitral area, probably depending upon some slight rheumatic alteration of the mitral valve. (4) The absence of a marked systolic murmur from the aortic area. (5) The loud diastolic murmur audible over all the cardiac area, beneath the level of the aortic orifice. And lastly, the markedly distinct character of both murmurs in all the arteries, especially in the carotid and femoral arteries. The absence of any distinct systolic murmur from the aortic area points to the impurity of the first sound in the mitral area being due to rheumatic alteration of the valves rather than to mere dilatation of the ventricle, and also indicates the absence of any well-marked obstruction at the aortic orifice; indicates, therefore, the preponderance of retraction or crumpling, or it may be of ulceration, over calcification of the valves. The absence of marked obstruction at the aortic orifice also accounts for the very free regurgitation indicated by the great collapse of the pulse, the distinctness of the (cardiac) diastolic murmur heard in the arteries, and also the distinctness of this murmur in the cardiac area. The latter, however, depends partly on the condition of the valve as it still exists, and its capacity for originating a fluid vein, though the force of the regurgitating current—largely depending on the freedom of regurgitation—must have its influence in making this fluid vein more sonorous. The condition of the sternum, and its connection with the ribs, in relation to its capacity for conducting sound, has also undoubtedly an important influence in propagating the murmur over the chest.

The points to which I wish particularly to direct your attention at present are what may be termed the specialities of this case: the absence of a systolic murmur from the aortic area, and the presence of a well-marked double murmur in all the arteries. I have already pointed out that the absence of a systolic murmur from the aortic area is due to the almost entire absence of any obstruction from the aortic



orifice. Some degree of obstruction is, however, present, the valve segments are not entirely obliterated, and to this it is due that the systolic murmur is more distinct in the innominate artery than it is in the aortic area. Some trifling fluid vein is formed at the aortic orifice, passes with the onward current and intensifies the other sources of murmur inherent in the artery itself. The systolic murmur is louder in the innominate artery than in the aortic area, but falls far short of that heard in the carotid artery, where compression with the stethoscope causes an independent fluid vein that increases and intensifies the murmur. Compression of the artery intensifies the murmur, but does not produce it; it is loud enough to be readily heard on placing the stethoscope merely in the neighbourhood of the artery. The systolic arterial murmur is composed of two elements, it is partly due to the fluid vein produced at the aortic orifice, but chiefly to the vibration of the arterial coats, caused by the sudden tension produced by the wave of blood, disproportionate to the arterial calibre, sent forward by the greatly dilated and hypertrophied left ventricle. Consequently in the carotid arteries, at the moment of the ventricular systole, a loud murmur is to be heard which is partly produced by a fluid vein originating at the aortic orifice, but in a much greater measure by vibrations caused by the sudden tension of the arterial coats.

A loud systolic murmur may thus be audible in the arteries in two very different conditions of the aortic orifice: *where there is much obstruction from calcified valves, the murmur is loudest at the arterial orifice, is propagated into the arteries and gradually dies away as we recede from the heart. Where there is great destruction of the valves, and consequently but little obstruction, the murmur is always less audible over the aortic orifice than over the arteries, and the diminution in intensity on receding from the heart is not so great.* In both cases the rhythm of the murmur alters as we approach the periphery, it is always in unison with the



rhythm of the pulse, and not with that of the heart. A ventricular-systolic murmur in the carotids may thus become ventricular-diastolic when it reaches the radial or femoral arteries; or if it still remain ventricular-systolic, then the cardiac systole with which it is in unison is not that which originated the murmur, but its immediate successor, as we may readily ascertain by tracing it down the arteries. *The diastolic murmur, however, which in the carotids immediately follows the ventricular-systolic murmur, is in every part of the arterial system synchronous with the arterial systole, whatever relation that may bear to the cardiac diastole, and has but one origin, viz., free regurgitation into the ventricle.* It is inaudible until we compress the artery with the stethoscope so as to cause the formation of a fluid vein at that point; then it is always to be heard with greater or less distinctness, according to the amount of regurgitation present. The readiness with which we hear this ventricular-diastolic murmur in the arteries may be received as a measure of the freedom of the regurgitation into the ventricle, but its absence is not to be accepted as a proof that no regurgitation is present, as, if much obstruction exists at the aortic orifice, this murmur may be quite inaudible above the aortic valves. I agree with Jaccoud<sup>1</sup> when he states that, on placing the stethoscope lightly over an artery at the moment of its diastole, only a slight membranous click is heard in the normal condition, synchronous with its sudden expansion. A stronger pressure may increase this sound, but it produces no distinct murmur. When, however, the artery is forcibly expanded by a large blood-wave coming from a dilated hypertrophous ventricle, the very slightest pressure converts this membranous sound into a loud systolic murmur. Should this condition be associated, as it frequently is, with free regurgitation, a slightly increased pressure brings out clearly enough a soft diastolic murmur immediately succeeding the systolic one, while a still stronger pressure extinguishes all

<sup>1</sup> *Leçons de Clinique Médicale* (Paris, 1869), p. 185.

murmurs and ultimately all pulsation. I agree also with Duroziez<sup>1</sup> in thinking this double arterial murmur to be in a great measure an artificially produced phenomenon, of which the elements certainly exist naturally in all suitable cases, but which can only be made audible by artificial means. But I differ from him entirely in his estimate of this double arterial murmur as an important sign in the diagnosis of aortic incompetence. In one respect I go further than Duroziez, for I have no hesitation in saying that a true ventricular-diastolic murmur is never heard in the arteries unless aortic incompetence exists.<sup>2</sup> But I have just as little hesitation in saying that aortic incompetence may exist in many cases in which no such murmur is to be heard. Traube<sup>3</sup> believes that when aortic regurgitation is extreme, we can hear in the femoral arteries a double sound—not a double murmur—and that this double sound originates spontaneously in the artery itself, without any intervention of the observer. He regards the systolic sound as produced by the vibrations of the arterial walls, caused by their great and sudden distention, and the diastolic sound as caused by their equally sudden relaxation. But the idea of a sound being produced by *sudden relaxation* of the arterial coats is so untenable from a physical point of view, that there is little wonder that the element of truth upon which Traube's theory was based has for a time escaped due recognition, especially as the phenomena concerned have no special relation to aortic incompetence, and are often well marked in widely different conditions. When we auscultate below Poupart's ligament, or at the root of the neck, or just below the clavicle, we can often hear one, two, or even three short, sharp sounds. In aortic regurgitation, one of these, always the second if two

<sup>1</sup> "Du Double Souffle Crural comme signe de l'Insuffisance Aortique," *Archives General de Medicine*, 1st April 1861.

<sup>2</sup> Duroziez says that this double murmur is also heard in cases of enteric fever, chlorosis, and lead poisoning.

<sup>3</sup> *Gesammelte Beiträge zur Pathologie und Physiologie* (Berlin, 1871), Bd. i. S. 793; and *Berliner klinische Wochenschrift* (1872), No. 48, S. 573.

sounds only are present, or the third if there be three, is the sound of arterial distention already referred to, and it is readily converted into a blowing murmur by pressure on the artery. The other sounds are heard most distinctly over the veins in the situations referred to, and no amount of pressure ever converts them into murmurs, even when that pressure can be effectively applied, as over the femoral vein. Pressure may extinguish these sounds, but it never transforms them into murmurs. These sounds are occasionally to be heard in aortic incompetence, at least as often in other forms of cardiac disease, as also in pulmonary affections, and they are always associated with tricuspid regurgitation. Friedreich<sup>1</sup> ascribes them, and I agree with him, to the sudden sharp closure of the venous valves, in the situations referred to, by the regurgitant blood-wave. When these phenomena are produced in the veins alone, the sound may be single or double; in the first instance it is produced by the ventricular systole alone, in the second, the first sound is due to the contraction of the auricle, the second to that of the ventricle. These venous sounds are to be heard in every form of disease of the heart or of the lungs which is capable of causing tricuspid regurgitation, but the arterial element only comes into play when we have to do with a case of aortic regurgitation. The sounds may then be double or triple; the arterial portion is always the last,—because the auricular contraction invariably comes before the systole of either ventricle; and the regurgitant wave from the right ventricle, favoured by the congested state of the veins, precedes the pulse-wave from the left ventricle, which is more than usually delayed by the altered physical condition of the arteries (*vide* p. 90). Naturally these sounds have not the significance ascribed to them by Traube; a double sound is not always to be heard when regurgitation is extreme, and it is often present when there is no aortic incompetence. Moreover, the venous sounds

<sup>1</sup> "Ueber Doppelton an der crural Arterie, sowie über Tonbildung an den crural Venen," *Deutsches Archiv für klinische Medizin*, Bd. xxi. S. 205.

cease to be heard as soon as the congestion is great enough to make the venous valves incompetent.

In the normal state of the heart and blood-vessels, the pulse-wave set agoing by ventricular contraction is generally believed to be arrested at the capillaries, but this is by no means always the case. Even in those who think themselves healthy, and whose condition certainly closely approximates the normal, the cardiac pulsation is occasionally propagated not only into the capillaries, but even into the veins, as was first recorded by Quinke. Such cases are, however, really abnormal, an abnormality due to failure of one or other of the three great factors of the circulation.<sup>1</sup> These are (1) the force and frequency of the heart's action, which must be sufficient to keep the arteries overfilled, that is to maintain the normal blood-pressure; (2) the peripheral resistance, that normal state of constriction of the arterioles called their tone, which helps to keep the arteries distended by preventing too rapid an outflow; and (3) the elasticity of the arterial coats, which when called into play by the two factors just described, converts the intermittent supply from the heart into the continuous flow of the capillaries and veins.

In aortic regurgitation the arteries are dilated, and their elasticity is greatly lost, and though the blood-pressure is not low in proportion to the freedom of regurgitation, yet the conditions are always favourable for the production of a capillary pulse-wave, which is more or less recognisable, and which is more marked and is more distinctly seen to pass into the veins the larger the ventricle and the more forcible its action. Hence a capillary or even a venous pulse—and now of course I speak of a direct venous pulse running up the arm, and not of mere jugular regurgitation—is frequently to be seen in cases of aortic incompetence, not only in inflamed or erysipelatous parts, but also in such parts as the nails, cheeks, and retina in their normal state. This capillary or direct venous pulse is always an important indication of

<sup>1</sup> Foster's *Physiology*, 3rd edition (1879), p. 136.



impairment of one or other of the three great factors of the circulation; it is frequently to be seen in aortic incompetence, but is by no means pathognomonic of that affection.<sup>1</sup>

From the very free regurgitation in Bailey's case he presented a well-marked example of all the peculiar phenomena just referred to. In the spring of 1875 he was again for several weeks under observation in Ward V. His cardiac phenomena remained unchanged, and he was again discharged much relieved. For one or two years Bailey returned to us during the cold weather of spring, and then ceased to appear. It is an interesting circumstance connected with his case that to the last he maintained himself as a coal porter, and was daily in the habit of carrying bags of coal to the upper stories of some of our highest houses. A very good example of the little breathlessness and discomfort that attend a compensated aortic incompetence, however free the regurgitation may be.

The next is the last case I shall comment upon at present, and I bring it before you now as an illustration of a third form in which aortic incompetence may occur.

CASE IV. Matthew Murray, one of our city police, aged thirty-nine, admitted to Bed 7, Ward V., on 19th March 1874, complaining of pain in the chest and breathlessness, from which he has suffered greatly since last October. He was originally a farm-servant, but has been in the police force for eleven years and a half. He is 5 feet 9 inches in height, stout, and healthy-looking; he has always been sober, well fed, clothed, and housed, and has never suffered from any serious disease—in particular, has never had rheumatism. Seven years ago he had sores on his penis, but they were not followed by any sore throat, or by any eruption, etc. In his earlier days he was in the habit of making violent and laborious exertions;

<sup>1</sup> *Vide* Mr. T. Wilkinson King, *Guy's Hospital Reports*, Nos. 4 and 12; Stokes on *Diseases of the Heart and Aorta* (Dublin, 1854), p. 202; Lebert, *Handbuch der practischen Medicin* (Tübingen, 1862), vol. i. S. 725; Quincke, "Beobachtungen über Capillär- und Venenpuls," *Berliner klinische Wochenschrift* (1868), No. 34, S. 357; Grandclement, "De la valeur des Battements de l'Artère Centrale de la Rétine dans les Affections Cardiaques," *Lyon Medical* (1874), No. 12, p. 136.



latterly he has not done so. He has never been in the army, and cross belts on the chest form no part of his official costume. About seven years ago he received a severe blow on the chest from one of the shafts of a lorry, while trying to stop a runaway horse. On inspection, marked pulsation is visible both in the carotid and brachial arteries; the cardiac pulsation is abnormal, no apex beat is to be seen, but there is slight pulsation in the *scrobiculus cordis*; both pupils are normal. On palpation, the radial pulses are felt to be equal, the pulse beats at the rate of 75 per minute, it is jerking and accompanied by a well-marked thrill. This thrill is not so perceptible in the carotids as in the brachial and radial arteries. The jerking character of the pulse is markedly increased on elevating the patient's arm above his head as he sits. The heart's apex is scarcely to be felt; it beats between the fifth and sixth ribs, three inches and a half from the left edge of the sternum. The heart's impulse is everywhere feeble. On percussion in the parasternal line, dulness begins at the upper edge of the third rib, and extends down to the liver dulness. Transversely in the line of the fourth rib, dulness commences half an inch from the right edge of the sternum, and extends across for a distance of six inches. On auscultation in the mitral area, the first sound is heard obscured by a systolic murmur, which is found to have its position of maximum intensity in the aortic area. This systolic murmur is heard to obscure the first sound in the mitral, in the tricuspid, and in the pulmonary areas; in the aortic area it entirely replaces it. In the aortic area there is no second sound, it is wholly replaced by a diastolic murmur, and this murmur replaces the second sound in all the areas except the pulmonary, where it greatly obscures an accentuated second that is only faintly to be heard. This double murmur is more or less audible over the whole chest, both before and behind, but it has its position of maximum intensity in the aortic area. The aortic dulness reaches to within one inch and a quarter of the top of the

sternum. A distinct double murmur is heard both in the carotid and in the femoral arteries. Dyspnoea and cough are chiefly remarkable when the patient lies down at night. Percussion over the left lung is normal, over the right lung it is slightly higher in pitch (emptier), especially over its lower lobe. Auscultation is normal over the left lung; over the right lung the vesicular murmur is weakened, especially over the lower lobe. In other respects the patient is normal.

After what has been already said, this case seems to require but little explanation, the mere recapitulation of the physical signs have revealed its nature, though to a tyro some may seem misleading. Thus the extent of the transverse dulness might lead one to suspect great dilatation of the right heart, but the very trifling character of the impulse in the *scrobiculus cordis* at once forbids this assumption. Again the free regurgitation through the aortic valve negatives the idea that the impulse between the fifth and sixth ribs is likely to be that of the true apex; while the forcible character of the blood-wave, as perceived in the arteries, is opposed to any idea that the feeble ventricular impulse is a true indication of the condition of the ventricular walls. But a little consideration leads at once to the conclusion that the increased breadth of the cardiac dulness is due to the oblique position of the heart, which lies more transversely and also deeper than usual, the defective impulse being due to the increased depth of lung covering the heart, and not to any preponderance of dilatation over hypertrophy. The cause of this pushing of the heart backwards and to the left must be sought for in some derangement of its normal relations, and a very efficient cause is to be found in the aneurysmal condition of the aorta. This vessel is not only largely dilated, as shown by its increased breadth of dulness, but it also has an aneurysmal bulging on its ascending portion just above the heart, pressing on the right bronchi, and chiefly upon that distributed to the lower lobe of the right lung, as indicated

by the rise in the percussion note specially marked over that lobe (*vide antea*, p. 20), as well as by the enfeeblement of the respiratory murmur over the corresponding part of the chest. In this case the aorta is probably in a state of cirroid aneurysm, largely dilated, with at least one bulging just above its origin. The case is given now merely as an example of a third mode in which aortic incompetence may be produced and its murmurs propagated. This patient has never suffered from rheumatism, and the incompetence of his aortic valves seems to be entirely due to dilatation of the aorta itself. As already stated (*vide antea*, p. 32), the first effect of dilatation of the first part of the aorta is to cause the aortic valves to close with unusual force, so that the second sound becomes markedly accentuated. As the dilatation increases, the valves become too narrow to close perfectly the enlarged orifice, and accompanying this accentuated second we have a diastolic murmur of greater or less intensity. Such an aorta is always more or less atheromatous, but unless there are projecting spiculæ the diastolic murmur is not generally audible above the valves; should the vessel, however, be roughened by projecting spiculæ, the diastolic murmur may commence high above the valves, and in any case it may be prolonged and loud enough completely to obscure the second sound. In the earlier stages of this affection, appropriate treatment not infrequently so favours the contraction of the vessel as to cause the complete disappearance of the diastolic murmur, but the second sound remains permanently accentuated, and as we may readily suppose, the diastolic murmur in such circumstances is exceedingly apt to recur. In all similar cases the valves are seldom much calcified, they are frequently quite flexible, and when this is the case they present no obstacle to the egress of the blood from the ventricle. Under these circumstances the systolic portion of the murmur is occasionally, though rarely, due to fluid veins formed by projecting spiculæ; more often to tensile vibrations produced in the arterial walls,

by the wave of blood disproportionate to their calibre sent forward by the dilated and hypertrophous ventricle ; but by far its most common and important cause is the production of fluid veins at the comparatively narrow orifice of the aorta, by the large blood-wave passing through it into the aneurysmal dilatation of the vessel beyond. If there be projecting spiculæ attached to the aortic walls, the diastolic murmur may be partly due to them ; it is always, however, mainly caused by the formation of fluid veins by the regurgitating blood at the aortic orifice, which forms a comparatively narrow strait between two parts of wider calibre—the dilated aorta and the ventricular cavity. This diastolic murmur has its position of maximum intensity at or below the aortic orifice, and owes its propagation over the cardiac area, and the chest generally, to the resonant qualities of the sternum and ribs. The double murmur in the arteries arises from causes similar to those which have been shown to have this result in the immediately preceding case, and has exactly the same significance. From the foregoing cases we learn, that amongst all the various signs which enable us to detect aortic incompetence or to predict its significance, the murmurs that accompany it, and their mode of propagation, are not the least important. And we must remember that though a diastolic murmur at or beneath the level of the aortic valves, in the line of the sternum, is pathognomonic of incompetence of the aortic valves, yet in regard to prognosis the mode in which this murmur is propagated is of more consequence than its mere detection.

The points of importance which have just been illustrated may be summarised as follows :—*A diastolic murmur at or below the level of the aortic valves, chiefly audible in the line of the sternum, is significant of aortic incompetence. If this diastolic murmur is inaudible in the carotid arteries, it is invariably accompanied by a systolic murmur, having its maximum intensity at the aortic valves or in the so-called aortic area, and this indicates comparatively trifling incom-*



*petence with considerable obstruction at the aortic orifice, most probably due to calcified semilunar valves.*

*If this diastolic murmur is distinctly audible in the carotid arteries, it is invariably preceded by a loud systolic murmur audible in these arteries, but not always to be heard in the aortic or in any part of the cardiac area, and this indicates very free regurgitation (considerable incompetence) with comparatively trifling obstruction. What has just been said in regard to the murmurs in the carotid arteries refers equally to murmurs audible in the femoral, as well as in all the other arteries. Each case differs from another; in practice we find the most marked examples of great regurgitation with slight obstruction, shading off into instances of great obstruction with but slight incompetence, and the murmurs indicative of these different lesions vary accordingly. The cases used as illustrative examples are fairly well-marked and pure instances of the three principal forms in which the murmurs of aortic incompetence present themselves.*

*A double murmur in the arteries is always the result of great incompetence. If associated with a history of rheumatism, this incompetence is due to great retraction and possibly ulceration of the semilunar valves, and accompanied by the absence, more or less complete, of the systolic murmur from the cardiac area. But if not associated with a distinct history of rheumatism, the systolic portion of the murmur will be well marked over all the cardiac area, and the lesion will be found to be arterial (aortic dilatation), and not primarily valvular in its origin.*



### LECTURE III

#### ON INCOMPETENCE OF THE AORTIC VALVE, WITH SPECIAL REFERENCE TO THE MODE IN WHICH THE DISEASE PRO- GRESSES, ITS PROGNOSIS, AND ITS TREATMENT

IN the previous lecture on Aortic Incompetence I have specially directed attention to the diagnostic significance of the murmurs accompanying it, because it is from these murmurs that we learn the probable character of the regurgitation present, a matter of some moment, as upon the comparative freedom of regurgitation, or the reverse, many of the consecutive phenomena and much of the prognosis depends.

When regurgitation takes place through the aortic valve, the left ventricle, during its diastole, fills from two sources instead of one—from the aorta as well as from the auricle. The first and most immediate effect of this is a sudden diminution of the arterial tension, which impresses its character on the pulse in exact proportion to the amount of regurgitation present. The second effect of the diastolic filling of the ventricle from two sources is, that when the systole of the auricle takes place, the left ventricle is over-distended, and the left auricle incompletely emptied. The result of this obstruction to the onward flow of the blood is passive congestion of the lungs, revealed, as such obstruction always is, by an accentuation of the pulmonary second. This passive congestion of the lungs, once started, must of course—if no compensating change were possible—go on

increasing with each cardiac pulsation. Ere long, in fact, *pari passu* with the pulmonary engorgement, the right ventricle gets over-distended, the tricuspid valve becomes incompetent, and the right auricle dilated, followed by passive congestion of the systemic veins, terminating in general dropsy, and a gradual lowering of the arterial blood-pressure till it falls below that compatible with life, and death ensues under symptoms of a gradually increasing asthenia.<sup>1</sup> This, in fact, is a common mode of dying in other forms of cardiac disease, though it is comparatively rare in aortic incompetence. Because the lesion (regurgitation) which in this affection obstructs the onward flow of the blood is able, under circumstances, to produce directly, and at once, such a fall in the blood-pressure as can only be brought about by other lesions indirectly, and after a long lapse of time.

In all cardiac lesions there is, however, a tendency to postpone this ultimate result by the production of secondary changes in the heart itself, which more or less perfectly compensate these lesions; and the great object of treatment is to promote this compensation, to make it as perfect as may be, and to maintain its efficiency as long as possible.

We often accidentally discover the existence of aortic incompetence in those apparently in perfect health, but they never come to consult us unless compensation is ruptured, and then the overloaded state of the heart gives rise to many distressing symptoms; such as breathlessness, uneasiness or pain in the cardiac region, with irritability of the heart itself, evinced by palpitation on the slightest provocation, direct or reflex, such as the most trifling exertion, or the mere ingestion of food. It is this ruptured compensation we have to remedy, not the aortic incompetence, which is beyond our skill; a

<sup>1</sup> "Systemic death consists in decline of aortic pressure. This decline may occur rapidly, as in syncope; but usually, even in deaths by violence, it is very gradual. In deaths from disease it may last (gradually decreasing) for days, weeks, or even months."—*Handbook for the Physiological Laboratory* (London, 1873), p. 209.

distinct remembrance of this will often be a great help to us in attaining the object we aim at.

When aortic incompetence is established, and the ventricle fills from two sources instead of one, the first effect is of course to overfill that organ, and the earliest attempt at compensation is the accommodation of the left ventricle to the increased amount of blood it is required to contain. This is readily brought about through the elasticity of the myocardium itself. The first step in compensation is thus dilatation of the left ventricle, but this only provides a receptacle for the blood delayed, and not a means of restoring it to the circulation. This dilatation itself must therefore be compensated before the balance of the circulation can be so efficiently restored as to make the lesion mute. But as the heart always works well within its powers, it has no difficulty in disposing of its fuller contents and sending on a larger blood-wave; this larger blood-wave distends the coronaries and thus provides an improved metabolism of the myocardium, so that *pari passu* with its dilatation the wall of the ventricle becomes thicker and stronger, it hypertrophies. In cases of disease in which regurgitation is only slowly developed this all takes place with so little disturbance of the heart or of the circulation generally, that it is often only after the lapse of many years, frequently passed in strenuous labour, that the patient learns to his astonishment that he has in all probability been for so long the subject of heart complaint.<sup>1</sup> On the other hand, if regurgitation has been suddenly developed in an extreme degree, as in the case of accidental rupture of one or more segments of the aortic valve, the immediate effect has occasionally been so great a disturbance of the circulation, that complete compensation

<sup>1</sup> I found this statement on the fact that I have seen many cases of aortic incompetence in whom the compensation had been accidentally, or unavoidably ruptured twenty years or more subsequent to one single attack of rheumatic fever, from which, in all probability, the lesion dated, coupled with my own knowledge that some have certainly suffered for nearly as long from aortic regurgitation without ever being conscious of it.

has never been established, the patient continuing to suffer till in no long time death has ensued.<sup>1</sup>

When aortic incompetence has developed as the result of local disease in a moderately healthy subject, in whom dilatation and hypertrophy of the left ventricle have gone on *pari passu*, so as to develop and maintain what is called perfect compensation for the lesion, a time at last arrives when this is no longer possible. Previous writers on this subject seem to have assumed that when this so-called compensation is once attained,—it may be after a longer or shorter period of cardiac perturbation,—it is maintained, accidents excepted, till nutritional changes in the myocardium render its longer continuance impossible.<sup>2</sup> But from this it appears that too much attention has been directed to the vital forces, and too little to the simple mechanical agencies involved in aortic incompetence, mechanical agencies which are, however, of vast importance in producing injury, and which have always to be considered and counteracted in all our therapeutic attempts to relieve the symptoms that accompany that lesion.

<sup>1</sup> Balthazar Foster, in his *Clinical Medicine*, p. 139, says that four years and a half covers the date of death in all such cases of traumatic lesion of the aortic valves. But I myself have seen, *post mortem*, aortic cusps which had been torn from their attachments, the lesion healed, and yet no serious symptoms developed till years subsequently. At this moment I am acquainted with a patient who more than twenty years ago had symptoms and signs apparently due to rupture of one of his aortic cusps, yet he still survives in much improved health. Experimental investigation is entirely at one with this experience, and quite opposed to the idea of such an accident being speedily fatal except under special and exceptional circumstances.

<sup>2</sup> Thus Jaccoud says:—"Quelque parfaite que soit la compensation des lésions cardiaques, elle est temporaire ; l'équilibre artificiel au moyen duquel elle maintient une circulation à peu près normale peut durer des années, mais il faut qu'il se rompe, cela est fatal ; le tissu musculaire du cœur atteint dans sa nutrition subit à la longue une transformation régressive, généralement grasseuse, et lorsque cette altération secondaire, dont Paget et Stokes ont montré toute l'importance, présente une certaine étendue, le ventricule, quoique augmenté de volume, quoique hypertrophié en apparence, ne contient plus assez d'éléments contractiles pour faire face au travail excessif que la lésion lui impose ; sa force propulsive diminue, il se vide mal, évacuation du système veineux est par suite gênée, la compensation est détruite, et comme cette rupture résulte d'une condition anatomique sur laquelle nous n'avons aucune prise il faut que le cœur s'arrête, et que le malade succombe."—*Leçons de Clinique Médicale* (Paris, 1869), p. 215.



The effect of a leakage, however trifling, once established between the segments of the aortic valve is, with the patient in the erect position, in accordance with Pascal's law,<sup>1</sup> that the interior of the left ventricle is constantly exposed to the dilating influence of a force equivalent to the weight of a column of blood the height of the top of the cranium above the heart, and of the diameter of the ventricular lumen. From the moment the leakage is once established till the cessation of life, this hydrostatic pressure never ceases, though it is modified by position: its action is not only continuous, but it increases, in accordance with the law referred to, *pari passu* with the gradual dilatation of the ventricle. All that the organism has to oppose to this powerful dilating force is first of all the natural elasticity of the myocardium itself, and second, the exercise of that well-known law whereby all hollow muscles hypertrophy and increase in strength in proportion to any obstacle opposed to the exercise of their function (Paget), provided their metabolism remains perfect. The danger to which such patients are exposed is thus very evident, as well as the manner in which any disturbance of the metabolism—general or local—may increase that danger. For example, if the system is in a depressed condition at the moment that aortic leakage is established, there may be a difficulty, or even an impossibility, of establishing compensatory hypertrophy, and the result may either be sudden death from asystole, or a greatly prolonged period of cardiac disturbance. Similar results may arise from similar causes at any moment during the persistence of the leakage, that is at any after-period of the patient's existence, and it is in the history of this lesion and its relation to cardiac metabolism that a great part of its interest lies.

If we regard compensation, once established, as perfect and complete, it is obvious that, apart from accidental disturbance,

<sup>1</sup> Pressure exerted anywhere upon a mass of liquid is transmitted undiminished in all directions, and acts with the same force on all equal surfaces, and in a direction at right angles to these surfaces.—*Traité de l'Equilibre des Liqueurs*, etc., par Blaise Pascal (Paris, 1763).



we must wait the development of local nutritional changes before we can have any disturbance of the compensation. If, however, we look upon compensation as always incomplete, though practically sufficient for the carrying on of the circulation, then a time must come when compensation is ruptured from purely physical causes, quite independent of any disturbance of either the general or local metabolism; and the latter seems the more correct view to take.

In aortic incompetence there are three possible conditions of the left ventricle, for it is to the state of that cavity that our inquiry in this matter is practically limited.

1. There is said to be occasionally a condition of over-compensation, in which the left ventricle is hypertrophied in excess of its requirements, and which is said to give rise to many disagreeable and possibly dangerous symptoms. This is a state of matters frequently referred to by many writers; it must, however, be one of extreme rarity, if it occur at all. It is a condition quite beyond my own experience, and one for which I can discover no rational explanation, and I am persuaded that for all practical purposes we can well afford to discard all consideration of any such hypothetical condition.

2. The balance of compensation may be perfect; the amount of compensation may be just sufficient to counter-balance the dilating effect of the regurgitation, and with a slightly dilated and hypertrophied heart the circulation may go on as before, till, from accidental or inevitable nutritional changes, compensation is ruptured, and disturbance of the circulation sets in.

3. We may have, from the moment that aortic leakage sets in, a dilating power acting on the left ventricle, which, from physical causes, must gradually increase in energy. This dilating force acts as a hindrance to the onward flow of the blood; the ventricle reacts against this obstacle, and for a time successfully, so far as maintaining the circulation is concerned, but always at a disadvantage. The dilatation

which commenced the organic disturbance continues in advance, the compensating hypertrophy which followed continues ever to lag behind, till at last a period is reached when asystole is threatened from physical causes alone, because the weight of the dilating fluid is in excess of the contractile force of the ventricle. At this moment, fortunately for the patient, the dilating force of the blood separates the segments of the mitral valve, and at the moment that ventricular paralysis is threatened by over-distention, the over-burdened ventricle is relieved by regurgitation into the left auricle, and the inevitable end is postponed. In the sequence of events just narrated every one must recognise the ordinary history of an uncomplicated case of aortic incompetence, and must also be fully aware that, even after the case has progressed so far, a patient may die with his ventricular muscle in a state of comparative health. We must all, therefore, be prepared to acknowledge the importance of Pascal's law in the sequential production of the organic changes consequent on aortic regurgitation, quite apart from any nutritional changes in the ventricle itself. But, though the hydrostatic influence developed by the incompetent condition of the aortic valve ultimately and inevitably gives rise to the condition just described, this may be precipitated by any general or local disturbance of nutrition. Whenever from general debility the metabolism of the left ventricle is interfered with, any sudden call for exertion or any violent emotion may be followed by asystole and sudden death even at a very early period of the affection ; or similar causes may produce over-distention of the ventricle and mitral regurgitation at a similarly unusually early period. This over-distention may be recovered from and fully compensated, but it marks a stage in the downward progress that is only too apt to be followed by an exceptionally early fatal termination. Similar results are threatened at any period of the disease when general nutrition is accidentally interfered with by privation, over-work, or any debilitating illness. In these circumstances the

disease ceases to be mute, cardiac disturbance sets in, and the patient comes under treatment. But the ruptured compensation may still be restored by appropriate management, and the patient may be reinstated in comparative health. It is otherwise, however, when the nutritional disturbance is local, and solely due to the progress of the disease. Judicious treatment may yet suffice to prolong life, but efficient compensation can no longer be restored, and though the end may be postponed, it is inevitable. The immense importance of a perfect metabolism to an organ doing so much work as the heart (equivalent, in its normal condition, to raising its own weight—10 oz.—13,860 feet high every hour) is self-evident, and we can readily understand how any impoverishment of the blood from age, privation, overwork, or disease, must impair the working power of the heart. And when we reflect that any impairment of the contractile force of the ventricle must tend to induce residual accumulation, and by the operation of physical laws still further to increase the obstacle with which the weakened ventricle has to contend, then we are able to form some idea of the difficulties with which such a heart has to cope, and the evils to which such a patient is exposed.

In order to have a clear understanding as to the manner in which aortic incompetence progresses, its probable duration, and its treatment, we must have a distinct understanding as to the physical agencies involved in that progression, and the mode in which the heart is enabled to resist them.

Now and then it happens that a morbid anatomist holding up a heart allows us to see water trickling through an aortic valve, the incompetence of which had not been detected during life. This need not give us any concern, because a certain force of stream is required to produce a fluid vein, and without a fluid vein there can be no murmur, and consequently no recognition of valvular incompetence. The crumpling of a rheumatic valve goes on so slowly that there is no reason to doubt that, in many cases at least, a

leakage between the segments of the valve has been long established before it attains force enough to reveal itself by a murmur. During all this time the leakage has been gradually increasing, and dilatation of the left ventricle, with its compensatory hypertrophy, have been equally slowly and gradually brought about through the agency of those physical and pathological laws formerly referred to,<sup>1</sup> so that we never hear a murmur of aortic incompetence without being at the same time able to detect some degree of eccentric hypertrophy of the left ventricle. When the incompetence is arterial in its origin, and depends primarily on dilatation of the aorta itself, accentuation of the aortic second often long precedes any other sign, and may even be associated with a systolic murmur of aortic origin for an indefinite period before the development of any sign of aortic patency, as has been pointed out by Stokes,<sup>2</sup> who has recognised and described these signs as early indications of a progressively advancing incompetence, which begins as slowly and as imperceptibly as in the rheumatic heart. Even when incompetence is suddenly developed by rupture of one of the aortic cusps, there is no reason to suppose that the accident is always attended by serious disturbance of the circulation. At least, I have repeatedly had occasion to observe cicatrices at the root of depressed and incompetent cusps, where there was no history of any sudden disturbance, and where circulatory troubles have only arisen after an indefinite period of calm following an accident, to which the rupture of the cusp seemed to have been due. It is true this is not always the case, and in some these troubles arise quickly and rapidly progress to their close, but this seems rather to depend on the previous state of the cardiac muscle than on the nature or position of the lesion. Rosenbach<sup>3</sup> has shown that when the aortic valves of

<sup>1</sup> *Vide antea*, p. 79.

<sup>2</sup> *Diseases of the Heart and Aorta* (Dublin, 1854), p. 227.

<sup>3</sup> "Ueber artificielle Herzklappenfehler," von Dr. Otto Rosenbach, *Archiv für experimentelle Pathologie und Pharmacologie* (Leipzig, 1878), S. 1.



healthy animals are suddenly destroyed there is no intermediate period, however short, of cardiac disturbance, but the heart at once adapts itself to its altered condition, without a trace of any fall in the arterial blood-pressure, such as must have been observed had there been but a momentary falter on the part of the heart. The dilatation<sup>1</sup> necessitated by the overfilling of the left ventricle from two sources, instead of as ordinarily from one, is provided for by the natural elasticity of the walls of the ventricle, and the increased power needed to expel the surplus blood is, at first, readily supplied by the reserve force of the heart, as that organ always has in health a considerable margin of reserve energy upon which the maintenance of life itself depends in many morbid conditions. But the extra large blood-wave sent out by the ventricle flushes the myocardium at a higher pressure, and thus aids the ventricle slowly to hypertrophy, under the influence of Paget's law, so that the reserve energy is from the first supplemented and ultimately fully replaced by an actual increase of muscular force. In this way dynamic compensation is for long efficiently maintained, though perfect structural compensation is never fully attained, the hypertrophy always lags slightly behind the dilatation.

It was formerly supposed that the heart could only be flushed with blood during its diastole, and by the agency of the arterial systole<sup>2</sup>—1st, because the blood-current during ventricular systole flows at right angles to the orifices of the coronary arteries, and only a small portion of

<sup>1</sup> Rosenbach errs in supposing that the dilatation in such a case differs in character from that occurring under other circumstances. He fancied that in aortic incompetence dilatation preceded hypertrophy, and that in other cases hypertrophy always preceded dilatation, *op. cit.* S. 12. But the only difference is one of degree, the mode of production is essentially the same under all circumstances.

<sup>2</sup> *Vide* Stroem, in Haller's *Elem. Physiol.* Vol. i. Lib. iv. S. 5, §§ 18, 19. (Lausanne, 1757); Thibesius, *Dissertatio med. inaug. de circulatione sanguinis in corde* (Lugd. Batavorum, 1708), §§ 24, 25; and Brücke, "Physiologische Bemerkungen über die Arteriae Coronariae Cordis," *Sitzb. d. k. k. Akad. der Wiss. zu Wien*, cl. Bd. xiv. (1854), S. 345.



the blood-wave could be supposed to find its way into them ; 2nd, because the myocardium during systole is so firmly contracted as still further to limit the quantity of blood getting into it at that time ; and 3rd, because in a considerable number of individuals the coronary arteries open so far within the valvular zone, that at the moment of ventricular systole their orifices are closed by the segments of the aortic valve thrown back on the arterial wall by the advancing blood-wave, so as completely to shut off any possibility of a systolic flushing of the cardiac muscle.

But Gaskell<sup>1</sup> has shown that even tetanic contraction of a muscle favours the arterial blood-flow through it, instead of obstructing it.

Hyrtl<sup>2</sup> has shown, from a special examination of 117 bodies, that in the greater number the orifices of both coronaries are above the free margin of the semi-lunar valves, and that one at least always is so. Hyrtl also states, as the result of numerous experiments on rabbits, cats, and dogs, that section of the coronary arteries is invariably followed by an intermittent blood-spurt coincident with the systole of the ventricle, and this he thinks conclusively proves that closure of the coronary opening by the segments of the aortic valve never occurs.<sup>3</sup> Moreover Ceradini has shown by a reference to the physics of the valve segments, as well as that of the blood-current, that the

<sup>1</sup> Ludwig's *Arbeiten* (1876) ; *Journal Anat. and Physiol.* xi. 360.

<sup>2</sup> "Beweis dass die Ursprünge der Coronar-Arterien während der Systole der Kammer von den semilunaren Klappen nicht bedeckt werden," u. s. w. *Sitzb. d. k. k. Akad. d. Wiss. zu Wien*, cl. Bd. xiv. S. 373.

<sup>3</sup> *Ueber die Selbststeuerung des Herzens, ein Beitrag zur Mechanik der Aorta Klappen* (Wien, 1855), S. 59, u. f. The self-regulative power of the heart, which means that the flushing of the coronaries is necessary for the diastolic expansion of the heart, is shown to be a myth by the fact that the heart of a frog or a shark will beat for hours even when altogether empty of blood. Panum and Von Bezold also found that the hearts of dogs and rabbits beat for hours after the coronary arteries had been completely blocked with wax. Vide *Virchow's Archiv*, Bd. xxv. (1862), § 308, etc., and *Untersuchungen aus dem physiologischen Laboratorium in Würzburg* (Erster Theil, 1867), § 256, etc. The error attached to Hyrtl's theory does not affect the truthfulness of his facts.

valve segments are not closely applied to the arterial wall during the ventricular systole, but float in the blood, maintained in equilibrium by the central or axial stream on the one hand, and on the other by reflux currents which originate at the exterior of the axial stream, flow outwards and downwards against the arterial wall, and are reflected from it upon the posterior surface of the segments of the aortic valve.<sup>1</sup> These facts seem conclusively to prove that the coronary arteries are patent to the blood-flow during the ventricular systole; the meaning of this being that the coronaries are flushed not merely by a reflux current of unknown value, but by a blood-current which, through the influence of Pascal's law, has the minimum aortic pressure of 200 mm. Hg. The cardiac muscle is thus freely supplied with highly oxygenated and nutritive blood at the full normal pressure at the very moment it most requires it, when the transformation of energy, from potential to kinetic, within its substance is at its height. This is in complete consonance with the fact that, except in very advanced cases of long standing, or where other causes have been at work, the heart in aortic incompetence is always found to be well-nourished and free from signs of degeneration. It also helps to explain the remarkable fact that sufferers from aortic incompetence are not necessarily short-lived; they are certainly liable to sudden death in a higher ratio than other men, but such patients often lead useful and wonderfully active lives quite unconscious of the existence of any serious disease. One of the most remarkable instances of this was a clergyman who had his first and only attack of rheumatism thirty-four years before I first saw him. His disease was then far advanced and only too surely approaching its termination, yet he assured me that till two or three months previously he had been perfectly unaware that anything

<sup>1</sup> *Der Mechanismus der halbmondförmigen Herzklappen* (Leipzig, 1872). Ceradini's work contains a very complete epitome of the literature of the subject.

ailed him. He was a married man with a family, and knew that all his life his heart had beat stronger than other hearts, but he regarded this as a sign of vigour rather than the reverse. Over-exertion at lawn-tennis precipitated the breakdown of his compensation, and this was too near its natural close to permit of recovery; he died three months after I first saw him. He was a man who discharged faithfully all the duties of his station, and took his share in all the amusements open to him, even indulging in such minor athletics as lawn tennis, curling, etc. I have seen many similar cases, some of them of even longer duration. Such patients are largely exempt from the troubles that afflict those labouring under mitral disease; the breathlessness, the frequent recurrence of dropsy, and of bronchitis, as well as the multiform uneasiness due to cardiac weakness, from which mitral patients suffer so much and so often, are largely unknown to those labouring under aortic incompetence. It is well it is so, as such accidents are of much more serious import in aortic than in mitral disease. The special immunity from cardiac symptoms enjoyed by those having well-compensated aortic incompetence has been noted by Niemeyer, who says that "such persons are frequently not even short of breath—a symptom never missed in mitral disease."<sup>1</sup> And he specially mentions a huntsman from Greifswald, who, though labouring under extensive stenosis and incompetence of the aortic valve, with immense eccentric hypertrophy of the left ventricle, was yet able to go through all the manœuvres and forced marches of the Franco-Prussian war without difficulty.<sup>2</sup> And this remarkable immunity from symptoms is readily understood when we consider the manner in which the heart is nourished, the mode in which compensation is established, and the fact that

<sup>1</sup> *A Text-book of Practical Medicine* (London, 1871), p. 347. This immunity of aortic patients from pulmonary symptoms is also referred to by Rosenstein, *vide* Ziemssen's *Cyclopedia of the Practice of Medicine* (London, 1876), vol. vi. p. 139, and by others.

<sup>2</sup> Niemeyer, *loc. cit.*

so long as the myocardium keeps healthy one of the best-nourished and most powerful muscles in the body prevents the development of any discomfort. Herein too lies the clue to successful treatment not only of aortic incompetence but of every form of cardiac failure : strengthen the myocardium by improving its metabolism and every unpleasant symptom will speedily vanish, but of that more anon.

This imperfect structural compensation long preserves its dynamic perfection, ever slowly advancing towards its natural termination when the heart has outgrown the feeding powers of its coronary arteries. Then the heart ceases to grow stronger ; and if its walls still grow thicker, this is due to venous congestion and the secondary development of fibrous tissue. The advancing dilatation is no longer closely followed by hypertrophy of the ventricle ; in no long time the segments of the mitral valve cease to come into apposition at the commencement of the ventricular systole, mitral regurgitation is gradually established, and though still liable to death from asystole, the natural termination of the disease is now by gradual asthenia, and is often preceded by considerable dropsy. The history of aortic incompetence is well fitted to awaken in our minds the highest respect for the recuperative power of the heart, even when it is irremediably injured. It ought also to impress upon all our minds the extreme unadvisability, to say the least of it, of treating a disease of this character, which may have only signs and no symptoms, as in such a case our duty is clearly to watch and wait, avoiding the *nimia diligentia*, yet prepared at any moment to act with energy and firmness.

The pulse of aortic incompetence is, as was first pointed out by Sir Dominic Corrigan, something entirely *sui generis*. In its most typical form it is large and full, quickly projected against the finger, and as rapidly falling off. Sometimes it communicates a vibratile jar to the finger. Scarcely has the shock of the impulse been perceived than the arterial tension drops to a minimum, from the aortic reflux, and the sensation



vanishes. This pulse—*pulsus celer et infrequens*—has been likened to the successive propulsion of shot against the finger, or to the jarring shock of the toy called a water-hammer. It is generally regular, and its characteristics are intensified by raising the extremity (arm or leg) in which we feel it perpendicular to the body as it lies horizontal, or by raising the arm above the head in the erect posture. But the characteristics of this form of pulse are subject to variations, and may be greatly modified by circumstances. Thus great obstruction, which also diminishes regurgitation, may lessen the distinctness of the jar very considerably, and this jar is often materially increased if much anæmia is present. A consecutive or coexisting mitral affection frequently impresses upon such a pulse the mitral characteristics of smallness, feebleness, and irregularity so strongly that its characteristics are entirely lost. Occasionally under these circumstances elevation of the arm brings out with tolerable distinctness the collapse so distinctive of aortic incompetence; but this is not always easily detected, and cannot be relied upon unless well marked. When it is distinctly recognisable, we are justified in assuming, from the twofold character of the pulse, the existence of a double lesion—aortic and mitral.

If we feel the apex beat of the heart and either of the radial pulses simultaneously in a perfectly healthy individual, it will be found that they do not beat together, but that the radial pulse lags behind that of the heart by a distinctly appreciable interval of time, which may vary slightly in each individual with special relation to the rapidity of the heart's action. But the radial pulse in health always precedes the second sound. This delay of the pulse is due to the fact that the arteries are elastic and not rigid, and that the blood moves along them as a wave, and not merely as a mass one end of which is extruded as the other intrudes. Even in health there are trifling variations in the rate at which the pulse-wave passes from the centre to the periphery, depending mainly on the blood-pressure and on the coefficient of the



elasticity of the arterial walls (the more rigid the vascular walls, the more rapidly the pulse is propagated).<sup>1</sup> In aortic regurgitation the dilated and hypertrophied left ventricle sends a larger blood-wave than usual into the arteries, and with greater force. From the forcible distention by this large blood-wave the arteries become more dilated and longer (more tortuous) than in health, and each pulsation is accompanied by greater arterial movement than usual (visible locomotive pulse). These phenomena are not, however, distinctive of aortic incompetence, as they may be due to other causes, though usually, in such cases, to a much less degree. In aortic incompetence the arteries are not only longer and more dilated than usual, but, from the reflux into the ventricle, they are also less distended; the blood-wave is thus longer of reaching its maximum, and the pulse is delayed. This delay of the pulse in aortic incompetence is invariable, as was first pointed out by the late Dr. Henderson,<sup>2</sup> the only apparent exceptions being those cases in which the delay is so great that the radial pulse coincides with the ventricular systole immediately succeeding that which has produced it. Any possible mistake in such a case may be prevented by tracing the blood-wave from the ventricle through the carotid and brachial artery to the radial,—not a difficult thing to do by the hand; while it may be both traced and timed with perfect accuracy by Brondgeest's Pansphygmograph,<sup>3</sup> or by the electric methods devised by M'Kendrick<sup>4</sup> and by Landois.<sup>5</sup>

Whenever, therefore, we have a visible, locomotive radial pulse,<sup>6</sup> which is delayed beyond the commencement of the

<sup>1</sup> *Vide Hermann's Handbuch der Physiologie* (Leipzig, 1880). Vierter Band, Erster Theil, S. 248; and Frey *Die Untersuchung der Pulse* (Berlin, 1892), S. 137.

<sup>2</sup> *Edinburgh Medical and Surgical Journal*, vol. xlviii. (1837), p. 369.

<sup>3</sup> Landois, *Lehrbuch der Physiologie* (Siebente Auflage, Wien und Leipzig, 1891), S. 130.

<sup>4</sup> *Edinburgh Medical Journal* (July 1874), p. 8.

<sup>5</sup> *Die Lehre vom Arterien-Puls*, von Dr. L. Landois (Berlin, 1872), S. 302, etc.

<sup>6</sup> Von Frey, *op. cit.* S. 235.

relaxation of that ventricular systole which originated it—as marked by the occurrence of the pulmonary second sound—then we have to do with an aortic regurgitation, which is probably great in proportion to the delay of the pulse.

The pulse of aortic incompetence is graphically represented by the accompanying figures (Figs. 7 and 8). In these tracings there is nothing to indicate undue delay of the pulse.

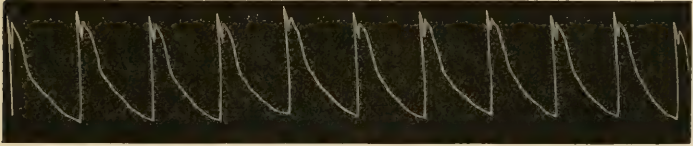


FIG. 7.

I have just pointed out how that is to be ascertained. Here we have the graphic representation of the local succession of events as they occur in an artery relaxed in all its dimensions, and more or less imperfectly filled. You will note the beak, or, as French observers call it, the *crochet*, at the summit of the abrupt line of ascent with which each tracing commences. This beak is due to the percussion-wave transmitted by the arterial wall outstripping the propulsion or blood-wave

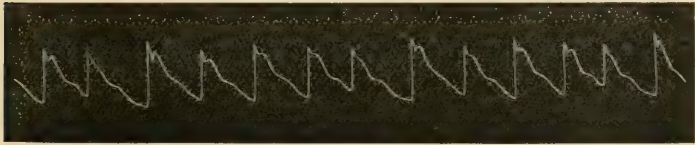


FIG. 8.

transmitted within the arterial lumen, and it may be increased, or more fully brought out, by taking the pulse-tracing with the arm elevated<sup>1</sup>; because, although in aortic regurgitation both kinds of waves are delayed, as a loose string vibrates more slowly and with larger undulations than one more tense, and an unfilled elastic tube is longer of reaching its maximum distention than one more fully distended, yet by the elevation of the arm the effect of gravitation on the blood increases the

<sup>1</sup> Loraine, *Sur le Pouls* (Paris, 1870), p. 258, etc.

delay the pressure-wave experiences in reaching the distal end of the vessel, while there is no additional obstacle opposed to the advance of the percussion-wave.

The comparative longevity, and the comparative frequency of sudden death in aortic and in mitral incompetence have been the subject of much debate and of much variance of opinion. These are questions which could only be decided by an appeal to statistics based on an accumulation of accurate data, involving the histories of many such patients, which unfortunately have yet to be collected. There can be little doubt, however, that the general impression is correct which assigns a shorter life to aortic than to mitral regurgitation. I have seen many cases of aortic incompetence who have been known to have suffered from that affection for thirty-five to forty years, but, on the other hand, I have also known many cases of mitral regurgitation who have suffered from their ailment for even longer, and one old friend, if his own statements could be trusted, and I believe they were trustworthy, had suffered from mitral regurgitation for close upon seventy years, and had lived an active and useful life for nearly all that time, nor was his heart the first to fail even at the last. Hospital patients, who belong to the labouring classes and have their compensation frequently disturbed by privation and overwork, have not so good a prospect of life under any circumstances, but even amongst them mitral regurgitation is always more favourable to life than aortic incompetence. Sufferers from mitral regurgitation often break down and return again and again to get rehabilitated; sufferers from aortic incompetence are not so fortunate, and, though there must be exceptions, four years reckoned from the day of admission will include the day of death in by far the larger proportion of those aortic cases who come under hospital treatment on account of serious disturbance of compensation. The limits of life in such cases may be said to lie between three months when serious rupture of the valve has occurred, and forty years in cases of

disease occurring under favourable circumstances, which may vary much in character. Within these limits our prognosis, always uncertain, must be determined by the circumstances of the patient, such as the necessity for manual exertion, his habits—temperate or otherwise, as well as any other conditions likely to influence his general health. Family longevity is also not to be discarded, as it affords a probable indication of a greater or less tendency to muscular degeneration. Further, the probable period during which the disease has already existed, the progress which it has already made, and the present condition of the cardiac muscle, all form important elements in relation to prognosis. Moreover, the temperament of the patient must not be neglected, as nervous excitability, whether revealed in sudden bursts of passion, or in irregular spurts of violent exertion, is always unfavourable, as whatever conduces to irregular action of the heart is injurious in all forms of cardiac disease, and is more frequently fatal in aortic incompetence than in any other.

Death from cardiac disease arises always—apart from accident—from syncope brought about in one of two ways, either by gradually increasing *asthenia*, in which the aortic blood-pressure slowly fails from day to day<sup>1</sup> till it drops at last below what is compatible with life, and death ensues; or by *asystole*, in which the blood-pressure suddenly falls below that necessary to maintain life because the heart ceases to act. No statistics are required to prove that the former accident is most likely to happen in mitral disease and the latter in aortic incompetence.

When the myocardium is enfeebled by inflammatory infiltration, fatty or fibrous degeneration, the heart may suddenly cease to act at any moment, either in systole or diastole—

<sup>1</sup> This is what seems to happen. But it may really be as when the ascending arch of the aorta in a dog is gradually narrowed by a screw-clamp, while the blood-pressure is recorded by a mercurial manometer connected with one of the carotids, that the blood-pressure does not actually fail till it suddenly drops just before death. *Vide* Roy and Adami, *British Medical Journal* (15th December 1888).



more often the latter—and with this cessation of action life comes to an end. Death from this cause may happen with or without valvular lesion. If any valvular lesion is present, there are, as we have seen, both anatomical and physiological reasons why it should be incomparably more frequent in connection with aortic incompetence than with any other valvular lesion. Even in the most extreme form of mitral stenosis, enough of blood always reaches the ventricle to enable life to be maintained, if only in a feeble fashion, while no obstacle is opposed to the free action of the ventricle. In free mitral regurgitation there is no want of blood in the ventricle, and provided the action of that organ is powerful enough, a sufficient quantity is always sent on to provide for the maintenance of life; and as the blood, in such cases, has always two ways of escape, ventricular systole, though it may be difficult, is always possible, except in the presence of muscular degeneration. In these two forms of disease, therefore, sudden death from asystole must be and is a somewhat rare accident; the more usual mode of death being syncope from asthenia brought about by secondary affections such as dropsy, with or without albuminuria, or jaundice, unless in those exceptional cases where death is hastened by sudden embolism of the brain or lungs, or by suffocation from an acute access of pulmonary oedema. On the other hand, in aortic incompetence the constant and continually increasing dilating pressure of the blood column acts as a permanent obstacle to ventricular contraction; and we can readily understand why comparatively trifling exertion, which raises the aortic blood-pressure, and also why emotional excitement, which through the vagus inhibits the action of the katabolic nerve, should in many cases be followed by sudden death. The inhibitory action, even with a comparatively healthy ventricle, permits the dilating force, already barely compensated, to turn the scale, and after a few feeble attempts to overcome the obstacle, the primary syncope passes into death from asystole, the heart remaining in a



permanent diastole.<sup>1</sup> If this be the case with a comparatively healthy ventricle, we can readily understand that it is much more readily brought about when the muscular tissue is diseased (fatty, fibrous, or both), as is so frequently the case. When the mitral valve has become incompetent from secondary dilatation, death from asystole is not so readily induced, though it is not altogether prevented. But as the congested cavity is no longer represented by the heart alone, but includes also the lungs, there is a possibility of relief to the oppressed heart by rupture of the turgid pulmonary capillaries. The relief thus obtained is mostly temporary, however, and only postpones the end. The mechanical cause always present in aortic incompetence, acts with continual increasing efficiency in the direction of one or other of these modes of death, and from the dilated and diseased condition of the pulmonary capillaries, and the consequent readiness with which they may be ruptured, we can readily understand that those cases of aortic incompetence which escape death from asystole mostly die from pulmonary apoplexy, or from a combination of the two. It is only rarely that dropsy and other secondary diseases of a serious character are established in such cases, death then occurring from asthenia. This is pretty much a question of animal mechanics, which statistics may confirm, but can never disprove. The manner in which death is caused, and the morbid appearances after death from asystole are precisely similar whether the end has come on the street, or at a public meeting, in the midst of apparent health, or after a longer or shorter period of more evident illness. In illustration of it I therefore prefer to give two cases in which the disease was known to have lasted for some time, because their history has an important bearing on the treatment of such cases.

CASE V. Isabella Stewart, aged thirty-five, employed in a

<sup>1</sup> This question has been very fully gone into by Dr. Mauriac. *Vide* his *Essai sur les Maladies de Cœur. De la Morte Subite dans l'Insuffisance de l'Aorte* (Paris, 1860); *vide* also *The Senile Heart*, by George W. Balfour, M.D. (A. and C. Black, London, 1894), pp. 31, 39, and 41.

paper-mill, admitted on 30th May 1870 to Ward XIII., complaining of cough, which troubled her for the first time about a fortnight ago, and of palpitation of the heart, from which she had suffered for about eighteen months. Her previous health had been good. Family history unimportant. Her expression was anxious, muscularity flabby, skin dusky, but naturally so. The lower extremities slightly œdematous, joints normal, she has never had rheumatism. Pulse 93, presenting in a well-marked form all the characteristics of the Corrigan or water-hammer pulse. On inspection and palpation the area of the cardiac impulse was found to be somewhat extended, the impulse itself feeble. There was visible pulsation of all the superficial arteries, particularly at the root of the neck, but this was by no means well marked. The apex of the heart was found to beat under cover of the sixth rib, at a distance of three inches and a half from the left edge of the sternum. At the level of the upper edge of the third rib, one inch from the left edge of the sternum, dulness commenced, and extended downwards to the liver dulness. At the level of the fourth rib, dulness commenced half an inch to the right of the sternum, and extended transversely across the chest for a distance of five inches. On auscultation over the apex, the first sound was heard much obscured by coarse crepitation, and rendered impure by the presence of a murmur, which, on being traced upwards, was found to have its position of maximum intensity at mid-sternum between the third and fourth ribs. The second sound is wholly replaced by a soft, diastolic murmur. This double murmur is audible with varying distinctness over the entire cardiac area; it is also propagated into the arteries, but the diastolic portion is only faintly heard in them. In the pulmonary region the pulmonic second is much obscured by the diastolic murmur, but on moving the stethoscope along the second interspace it is distinctly audible about an inch to the left of its usual position. The respirations are 24 per minute; the cough troublesome; sputa thin, watery,

and somewhat frothy, amounting to about 3 oz. in two hours. The pulmonary percussion is everywhere normal. On auscultation the normal respiratory sounds are entirely obscured by crepitation, cooing, and sonorous rhonchi. Appetite gone; some thirst; urinary and other systems normal. *Diagnosis*—acute bronchitis engrafted on and aggravated by an œdematous and congested condition of both lungs, the result of aortic incompetence. *Prognosis*—grave, death being threatened by cardiac asystole. *Treatment*—full diet, 6 oz. of wine daily, dry cupping to the chest, with a large jacket poultice afterwards, and a mixture containing digitalis, carbonate of ammonia, iodide of potass, and tincture of hyoscyamus, every four hours. It is noted that up to 15th June the patient's condition had been repeatedly aggravated by attacks of pulmonary congestion and threatened asystole, from which she had been relieved by dry cupping, stimulants, and the use of the mixture prescribed. On 15th June there was almost no cough, the expectoration was nearly gone, vesicular respiration everywhere audible; pulse 88, full and strong; patient was lying quietly, and expressed herself as quite relieved. On 18th June she was up at visit, and was apparently quite well. On the 22nd she had another attack of pulmonary congestion, with threatened asystole; from this she was temporarily relieved by treatment, but she died suddenly on 26th June. At the *post-mortem* examination of the body, which took place fifty-six hours after death, the heart was found more dilated than hypertrophied; it weighed 19 oz.; both ventricles were filled with dark-coloured clots, of which that on the left side extended some distance up the aorta, presenting one continuous mould of the interior of both ventricle and aorta. The aorta itself was slightly dilated, its valves thickened and incompetent, a considerable angular aperture being left between the edges of its cusps. The pulmonary, mitral, and tricuspid valves were healthy and competent. A portion of the muscular substance of the

heart was somewhat inflamed.<sup>1</sup> The right lung was healthy in appearance, and also on section, except at its lower margin, where there was a patch of pulmonary apoplexy. The pulmonic glands were infiltrated with black pigment, and there was a good deal of pigment on the surface of the lung. The left lung was adherent all round to the chest wall, especially posteriorly, the connecting lymph was recent, and in some places quite one-fourth of an inch in thickness. The lung itself was œdematous, and the bronchial mucous membrane congested. Iodine produced a slight mahogany tint on the cut surface of the liver, which was fatty, and presented a nutmeg appearance. The spleen was healthy, the kidneys congested, but otherwise healthy; the Malpighian bodies were slightly prominent, but gave no reaction with iodine.

This case presents a well-marked example of death from asystole, in which compensation had never been fully developed, and in which death was hastened by inflammatory complications.

In the following case asystole was the result of long-continued disease, which had been fully compensated, but in which the compensation had been accidentally ruptured. Even in this case, therefore, death was a preventable accident, though the unavoidable end was not long forestalled.

CASE VI. William Yorkston, a blacksmith, aged fifty-four, admitted to Ward V., 14th November 1870, complaining of orthopnœa, cough, expectoration, occasional pain in the chest, and swelling of the legs. About six weeks ago he caught cold after prolonged exposure to heat at his work, and this cold was aggravated by fresh exposure about a fortnight ago. He has hitherto enjoyed good health, and has never had

<sup>1</sup> This is all that is noted in regard to this matter in the *Pathological Records*:—Myocarditis is always a grave complication in serious valvular lesions; unfortunately, we cannot detect it with certainty, and are seldom even in a position to suspect its existence. I shall subsequently have occasion to relate a case of aortic incompetence in which myocarditis had an undoubted influence in promoting fatal asystole.



rheumatism; but for the last three or four years he has been working hard and drinking heavily. His father and mother both died of cholera. The patient was tall and muscular, his expression anxious, his face pale, his lips livid, his skin and joints normal, his legs œdematous up as far as the knees. The radial pulse jerked slightly, but not in any noticeable way; it beat 90 per minute. The jugular veins pulsated visibly; the right carotid slightly, the left carotid not visibly. The cardiac impulse was diffuse and feeble; the apex beat diffusedly between the sixth and seventh ribs. At one inch from the left edge of the sternum, dulness on percussion commenced at the upper border of the third rib and extended down to the liver dulness. Transversely on the line of the fourth rib, dulness commenced half an inch to the right of the sternum, and extended across for a distance of five and a half inches. In the mitral area a murmur was heard replacing the first sound; a second sound was audible. On tracing the systolic murmur upwards along a line joining the mitral and aortic areas, it was heard to grow gradually fainter as it neared the sternum, and it became again gradually louder as it approached the sternal end of the second rib on the right, where it attained a second maximum. In this position the second sound was wholly replaced by a loud but soft diastolic murmur; the systolic portion of the murmur alone was propagated into the carotids. In the pulmonary area a second sound was audible obscured by the diastolic murmur; it became more distinct when the stethoscope was moved off the sternum along the second interspace. At the lower end of the sternum the systolic murmur was very loud and distinct, and wholly replaced the normal sound of the tricuspid. The respirations were 26 per minute; cough hard and frequent; sputum watery, frothy, and small in quantity, no blood. The percussion of the lungs was normal. On auscultation sonorous and sibilant rhonchi were heard over the whole chest, partly obscuring the vesicular breathing. Appetite defective, bowels costive, urine scanty, no albumin



Patient slept none at night. He was ordered full diet and 4 oz. of whisky daily.

R Tincturæ scillæ,  
Tincturæ digitalis, ā ā ʒ ii  
Aquæ Cassiæ, ʒ vss. Misce.

Signetur—One tablespoonful every four hours.

R Elaterii gr.  $\frac{1}{8}$  fiat pilula—One such to be taken every four hours till the bowels are freely moved. To have 20 grains of chloral every night at bed-time.

On the 21st of November it is noted that the patient slept the previous night for the first time in the recumbent posture. His dyspnœa was comparatively trifling, the cough gone, the respiration free, the œdema of the legs entirely gone, the cardiac impulse stronger, the pulse more markedly jerking, only 80 per minute. The radial pulse is delayed for the full half of a cardiac cycle beyond the apex beat; the superficial arteries pulsate now in a marked manner, the jugular veins less distinctly. Over the mitral and tricuspid areas the systolic murmur is not so loud as formerly, and cannot be so distinctly localised. He was kept some time under observation, and then discharged much improved. On the 18th of August 1871 he was readmitted, and died suddenly next day. At the *post-mortem* examination the body was found to be well developed; skin and conjunctivæ of a yellowish colour. The heart was greatly enlarged, dilated and hypertrophied, weighing thirty ounces. Both ventricles contained large black clots. The aortic valves were atheromatous and incompetent; the aorta itself was greatly dilated. The mitral and tricuspid openings both admitted five fingers. The right lung was very dark in colour, greatly congested, and apoplectic; it weighed 2 lbs. 2 oz. The left lung also contained an apoplectic clot, and was covered with recent lymph. The liver weighed 3 lbs.; it was fatty, and its ducts congested and obstructed by catarrhal mucus. The kidneys were enlarged and congested.

These two cases illustrate very well the condition of the

heart found after death from asystole in aortic incompetence. Both ventricles in diastole, and both, but especially the left one, distended by a large black clot extending into the aorta. It appears as if death in these cases was due to paralysis of the heart from over-distention, which, as we have seen, may be brought about in various ways, and is that mode of dying in which death primarily invades the heart in aortic incompetence. One great object of our treatment of such cases is to prevent this mode of death which is continually threatened, and in obviating this primary danger we are also able, in a great measure, to prevent many of the secondary causes of death, or at all events to delay their fatal result.

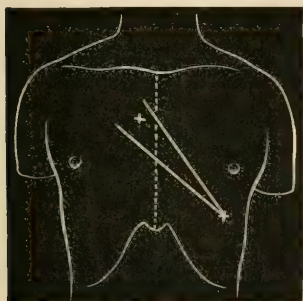


FIG. 9.

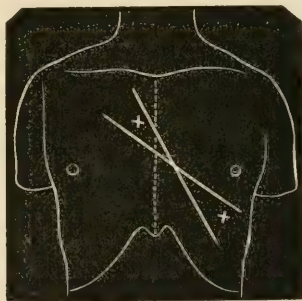


FIG. 10.

In Case V. it is noted that the first sound over the mitral area was obscured by a systolic murmur, which had its focus of maximum intensity in the aortic area. This we ascertained by tracing the murmur upwards along a line joining the mitral and aortic areas. The result is graphically shown in the accompanying diagram (Fig. 9).

In Case VI. the systolic murmur is stated to have had two foci of maximum intensity. In tracing this murmur upwards along a line joining the mitral and aortic areas, it was found gradually to decrease in intensity as we approached the sternum, and it again became louder and more distinct as we got nearer to the aortic area. This is graphically represented in the accompanying diagram (Fig. 10).

This is the method to be pursued when we wish to determine whether we have to do with a systolic murmur of mitral or of aortic origin, or with a systolic murmur of twofold origin—arising at both orifices. These diagrams represent graphically the method we employ and the result obtained in two out of the three hypothetical cases. When the systolic murmur is of purely mitral origin, its graphic representation is of course exactly the reverse of that depicted in Fig. 9.

In the treatment of aortic incompetence, we must carefully distinguish between those in whom the disease is only to be recognised by signs, and those in whom it is revealed by symptoms. In the former class the affection is dynamically compensated and is mute; in the latter the compensation is incomplete or ruptured. Those in whom the affection is only revealed by signs require no special treatment. Such patients or their friends must be cautioned against over-excitement or any sudden or violent exertion; they should be well fed, protected as far as possible from cold or any risk of chill, and stimulants should either be altogether avoided, or only used *pro re nata*, as their constant use has a tendency to weaken the myocardium. Apart from these cautions the patient must be left pretty much to his own devices, as continual watching and coddling, while it embitters life, yet affords no perfect security against the occurrence of sudden asystole, which is the only risk the patient runs at this stage of his disease—a risk moreover which is by no means certain to emerge even though a diametrically opposite course of conduct be pursued from that just recommended. A knowledge of this, a result of the great recuperative power of the heart, will prevent us from making the life of any one miserable merely because he happens to have a murmur of aortic regurgitation, and will enable us to view with comparative equanimity many departures from the strict laws of prudence on the part of those so affected. Such patients have generally good health, are quite capable of

enjoying life, and have no need of any special treatment. Iron, strychnine, and arsenic are useful tonics, when such remedies seem to be indicated, and we ought always, in such cases, to avoid any perturbative treatment likely to depress the vital powers.

When, however, compensation is ruptured and symptoms are superadded to signs, then we require to act more energetically; and should the disease be not too far advanced, our energetic action will often be followed by the happiest results, compensation will be restored, and life prolonged, sometimes for many years.

In aortic incompetence a most injurious effect is produced by the dilating force of the column of arterial blood, which, no longer resting upon the closed aortic valve, acts as a dilating force within the ventricle, according to Pascal's law, in proportion to its height and the area of its basis, and our primary object is to reduce as far as possible both of these elements. First of all, therefore, we put the patient to bed and endeavour to get him as nearly recumbent as possible, so as to diminish the height, and thus lessen the distending power, of the arterial column. With this view we deaden his sensibility by the administration of chloroform, chloral, or morphia, either by the mouth or hypodermically. The orthopnoea, which is so distressing a feature of this disease, has reference solely to the congested and œdematous condition of the lungs, and is established by nature to give the patient's respiratory muscles a better purchase in elevating the chest walls. A wise physician knows that whatever risk may underlie the condition of the lungs, there is much greater and more immediate danger connected with the state of the heart. He does not argue the point with nature, but he calms her instinctive fears with the means at his command, and feels he has gained a victory for science and a benefit for his patient when he has got him fairly recumbent. The inexorable mechanics of the circulation are appeased, and we have leisure to attend to the physiological part of the diffi-



culty. So intricately involved, however, are all our vital actions that we cannot appease the mechanical part of the difficulty without advantage to the physiological part, and *vice versa* we cannot improve the physiological condition of the heart without simultaneously appeasing its mechanical difficulty. It is advantageous, therefore, to carry out both parts of our treatment at once. And though this may look like an empirical treatment of symptoms, a deeper knowledge of the mechanics and physiology of an aortic heart, and of their mutual interdependence, shows that we are really employing scientific means according to scientific method, and that we apparently thwart nature only the quicker to secure the benefits she is aiming at. While, therefore, we endeavour to get the patient into the recumbent posture, so as to lower the height of the distending column and thus at once relieve the cardiac circulation and diminish the pulmonary congestion, we also endeavour to secure a similar relief by diminishing the area of the base of the arterial column, by contracting the heart and especially the left ventricle. So far as our present therapeutical armamentarium extends, there is only one drug by which this contraction may be certainly and safely secured, and that is digitalis. It is not so long since digitalis was regarded as the opium of the heart; now we know that it acts by increasing muscular elasticity, especially that of the myocardium and of the muscles of the arterioles, and by improving the metabolism of the myocardium.<sup>1</sup> It is still regarded by many as not only useless but dangerous in aortic incompetence, because it slows the heart's action and by prolonging the diastole gives longer time for regurgitation, and is supposed thereby to promote over-distention of the left ventricle and to increase the tendency to asystole, that very accident which digitalis is the most powerful agent we can employ to prevent. We know that the prolonged diastole in bradycardia promotes

<sup>1</sup> Vide *New Official Remedies*, by Ralph Stockman, M.D. (London, 1891), p. 58; also Lecture XIV., "On the Therapeutics of Cardiac Disease."



distention of the cardiac cavities, and that in these cases attacks of syncope, if not of asystole, are common enough. But in bradycardia the heart is filled by the blood in the course of its onward flow, and, as the heart's action is often slowed down to 40, 30, or even less than 20 beats per minute, the blood has often more than double the normal diastolic pause during which to distend the heart. In aortic incompetence the pulse is ordinarily much quicker than usual. Corrigan reckons it as averaging 110 per minute,<sup>1</sup> and it is neither necessary nor desirable to slow it down by any treatment much below the normal (say to 70). In fact in the case just narrated all the benefit desired was obtained without the pulse-rate falling below 80. There is thus no question of any abnormal prolongation of the diastole, and to suppose that to reduce the pulse-rate to normal is to induce a dangerous prolongation of the diastole, is to presuppose aortic incompetence to be much more dangerous to life than it really is, and to regard the danger as greater the more normal the rate of the circulation. The dynamics of the heart involve many problems which have not yet been solved, and one of the most interesting of these is the effect of prolongation of the diastole upon regurgitation through an incompetent aortic valve. One or two questions connected with this subject I am able to answer: Does undue prolongation of the diastole promote the occurrence of asystole? Never in my experience. Is asystole the result of over-distention, or is over-distention the consequence of asystole? In all the cases of aortic incompetence which I have seen terminated by sudden asystole, this has always seemed to have been brought about by some sudden interference (emotional or otherwise) with the action of the katabolic nerve, and the over-distention has seemed to follow and not to precede this interference. For all therapeutic purposes it seems sufficient to know that it is never necessary to prolong the diastolic pause beyond the normal to obtain all the benefits which digitalis is capable of bestowing,

<sup>1</sup> *Edinburgh Medical and Surgical Journal* (April, 1832), p. 241.

and to add that never in the course of a long experience have I ever seen any harm follow the use of digitalis, while I have many a time seen the greatest benefit follow the employment of that drug. As an indication of the nature of these benefits I may give the following quotation from a letter sent me by a patient, himself a highly educated medical man, a professor in one of our Northern Universities, who had long suffered from aortic incompetence: "When first I saw you my pulse was irregular and feeble. In the morning I could only walk about 250 yards. This took me from nine to twelve minutes, and I had to pause to gain breath three or four times on each occasion. In the afternoon, with usually the assistance of an arm, I could walk half a mile, resting say three times, but being troubled with occasional dull pain in the heart, constant distress from a feeling of constriction in the lungs, and a feeling of strain and weakness in the muscles. My stride was little more than half its natural length; and this half mile took me always over half an hour to accomplish. The night before I took the digitalis was the most trying and distressing which I can remember. My breathing was just a succession of rapid gasps, which ceased whenever I seemed about to fall asleep, when I had to draw a breath by an effort of will. The inhaled air did not seem to get fully into the chest. There was mental confusion, if not absolute cowardice. The feebleness and irregularity of the pulse were extreme, and my condition was altogether so painful that a sense of duty alone restrained me from desiring death. I had to walk all night till five in the morning. After the first day of taking digitalis all this was for the most part changed. Even then, as I expressed myself to you, I was 'a different man.' I think any one might be satisfied who has a pulse like mine now; I sleep well, the stride has lengthened out, five to six minutes now suffice for the 250 yards; with sometimes no rest at all I have walked about a mile and a half. Though there is occasionally a little panting, the air seems to get to the bottom of my chest, which is altogether free from any

feeling of constriction. In fact for a week I have been enjoying life ; there is a kick in my pulse which tells of everything firming up, and there is not in my mind an atom of doubt as to the *post hoc propter hoc* in my case."

The action of digitalis on the myocardium is to increase its elasticity and to improve its metabolism ; it is therefore the tonic of all others most useful in aortic incompetence, as it enables the myocardium not only better to withstand the dilating influence of the blood-column, but also to overcome its inertia with greater vigour. This increased elasticity lessens the distention of the heart, and contracts its volume without increasing its hypertrophy, while the improved metabolism of the myocardium increases its contractile force, and enables it to maintain the equilibrium of the circulation. Digitalis has no influence in itself in promoting hypertrophy, that is the result of an organ "being more than usually exercised in its office,"<sup>1</sup> and is the ultimate and necessary effect of the increased amount of work thrown on the ventricle by the incompetence of the aortic valve. By improving the elasticity and increasing the vigour of the muscle, digitalis rather retards than promotes the need for any muscular overgrowth. Hence digitalis is a drug which may be given in moderate doses at any period of the disease, not only without harm, but with positive benefit. Still it is a remedy not specially indicated so long as the affection is only revealed by signs alone and not by symptoms also. With a heart failing from any cause (ruptured compensation) it is different ; then digitalis is urgently called for and may be freely given with the utmost benefit. As the elasticity of the myocardium in an aortic heart has to be restored in the face of a gradually increasing strain, a large dose is always required, and in my experience has always been well borne. In the case just referred to the dose given was half an ounce of the infusion of the Edinburgh Pharmacopœia (equivalent to gr. iii. of the powdered leaves) night and morning for many weeks, with

<sup>1</sup> Paget's *Surgical Pathology* (London, 1870), p. 49.

nothing but increasing benefit. This may be reckoned about an average dose for such cases, and it may be increased or diminished according to circumstances. In one remarkable case of persistently threatened asystole, I was only able to get the patient removed from the infirmary, and taken home a distance of a hundred miles, by the continuous administration of half-drachm doses of the tincture of digitalis every two hours for several days.<sup>1</sup> This was, by the way, a case in which asystole ought to have been precipitated instead of being averted by the treatment, if the fears of Corrigan and his followers had any foundation in truth. This patient was not only able to be taken home in safety, but she lived for some time afterwards. Naturally in giving such large doses it is needful to keep a watchful eye upon the patient, though I have never seen the development of any untoward symptoms in any such case. Aortic cases not only require large doses, but they also seem to bear them well. The pallor of the countenance so common in aortic incompetence is greatly more due to defective circulation than to mere want of iron in the blood, but iron is always a good tonic in such cases, though it should not be given along with digitalis, as the combination is extremely apt to induce sickness. Arsenic may be very beneficially combined with digitalis; it is an excellent cardiac tonic, and its effects in relieving anginous pain, especially that form of it associated with diseased aortic valves, is often marvellous. Arsenic is perhaps more often conjoined with strychnine than with digitalis, as these two are most excellent tonics for ordinary use, the digitalis being only specially required when the compensation is ruptured. All such patients require to be nutritiously yet moderately fed, as the defective aortic pressure reacts injuriously on both the gastric and hepatic secretions, limiting both their supply and efficiency. Alcoholic stimulants, in moderation, are often useful in aortic cases, but their use must be watched and

<sup>1</sup> Agnes Butler, admitted to Ward XIII., 19th October, discharged 16th November 1872.

regulated. They are chiefly of value in helping to tide over a period of weakness, and have neither the special advantages of digitalis nor the important tonic effects of arsenic and strychnine.

When there is much starting from sleep, the *subitanea excitatio e somno* of the older writers, it generally dies away in a few days under the use of digitalis, and is much relieved by the use of morphia, though it does not always require it. Now and then cases of aortic incompetence are associated with mental aberration of a more or less violent character; there can be no fixed rule of treatment for such aberration, which must depend upon its degree, its character, and its cause. In several cases which have occurred to myself, and in which cortical anæmia seemed to be the chief exciting cause, full doses of morphia hypodermically, with digitalis, and a nutritious dietary were attended by the happiest results.



## LECTURE IV

### ON THE MURMURS AND OTHER PHYSICAL SIGNS DISTINCTIVE OF MITRAL STENOSIS

IF asked to state the sign that is supposed to be most distinctive of disease of the mitral valve, probably nine persons out of ten would without hesitation reply, a systolic murmur loudest at the apex. Yet this belief, though a common one, is far from being true. A systolic apex murmur is by no means a proof of any positive derangement of the cardiac mechanism. A murmur in this position may be of exocardiac origin, and the valves in this case may be perfectly healthy, and their action normal, notwithstanding the persistence of a murmur with the characters described. It is even alleged that a murmur of this kind may be of endocardiac origin, the valvular mechanism remaining intact; and though such a murmur must be very problematical, it is yet a possibility we cannot altogether ignore in estimating the probabilities in favour of our diagnosis. But systolic apex murmurs of exocardiac, or even of endocardiac origin, unaccompanied by valvular derangement, are not of course associated with regurgitation into the auricle, and are therefore free from any of the signs and symptoms associated with that accident. Moreover unequivocal proof that regurgitation does exist along with a systolic apex murmur is not a certain sign that disease or deformity of the mitral valve is actually present, because in a considerable proportion of those cases in which both of these phenomena occur the mitral valve is perfectly healthy. In fact the mitral valve may be free from

disease, and the auriculo-ventricular opening perfectly natural and undilated, and yet regurgitation may, and often does occur. How this may be I shall take another opportunity of explaining,<sup>1</sup> for the present it must suffice to state that even when mitral regurgitation is clearly established, this is no certain proof that the valve is diseased. On the other hand, there is a murmur so invariably associated with disease of the mitral valve that, when once heard, it may be accepted as a positive proof of a permanent deformity of that valve, and that even although the murmur itself may cease to be heard, as not infrequently happens. This murmur, so truly pathognomonic of mitral disease, has been termed, *par excellence*, the presystolic murmur, because it precedes any external manifestation of the cardiac systole. Nevertheless it is a direct murmur, occurring in the course of the onward flow of the blood, and produced by the contraction of the auricle which a glance at the diagram (Fig. 4) on page 39 shows comes at the end of the long pause and is the actual commencement of the cardiac cycle.

The history of the presystolic murmur commences with M. Fauvel,<sup>2</sup> who, in 1843, not only described its characteristics as a loud, rough murmur, running up to and terminating with the first sound, *un bruit de rape intense, précédant le premier bruit, finissant avec lui*—but also represented it as pathognomonic of stenosis of the mitral opening, and gave it the name by which it is best known among us—presystolic—because he recognised that it immediately preceded the ventricular systole. He also showed that this murmur really occupied the time of the auricular systole, and was due to the friction of the blood forced through a constricted mitral opening.

<sup>1</sup> Vide Lecture VI. "On Curable Mitral Regurgitation."

<sup>2</sup> *Archives Générales de Médecine*, 4me série, tome i. p. 1; vide also "The Evolution of Cardiac Diagnosis from Harvey's days till now," by George W. Balfour, M.D., *Ed. Med. Jour.* (June 1887), p. 1076. Fauvel borrowed the term presystolic from Gendrin, but Gendrin used it in a different sense; he applied it to the latter half of the long pause, terminating before the auricular contraction, which he rightly made part of the systole. Vide *Leçons sur les maladies du Cœur* (Paris, 1841), tome i. p. 111.

British physicians are, however, mainly indebted for a clear understanding of this important murmur to the lucid descriptions and instructive diagrams of Professor Gairdner of Glasgow, first published in 1861. Gairdner gave to this murmur the new name of auricular-systolic, expressing by this happy combination both its mechanism and its rhythm. There is every reason to believe that this most expressive and distinctive epithet helped to fix in the minds of observers the chief points connected with this murmur, and by so doing hastened its recognition in this country. In France, on the other hand, this recognition seems to have been delayed partly by the division of the cardiac cycle into a *premier temps*, including the systole of both auricles and ventricles, and a *second temps*, including the diastole of both these organs, and partly by Fauvel's adoption of the perfectly inconsequential term presystolic, a term that had been already employed by Gendrin in a more easily comprehensible sense.

In estimating the nature of any murmur supposed to be of valvular origin, there are two facts in regard to which we must be absolutely precise : the first of these is the position of maximum intensity of the murmur on the cardiac area ; and the second is its rhythm—that is its exact relation to the several physiological acts which constitute a cardiac cycle. These points being determined, the ascertaining of the lesion upon which the murmur depends is a simple matter of ratiocination in which it seems hardly possible to err.

The presystolic murmur—as we may now call it for shortness sake, without any fear of misapprehension—has a fixed and determinate position within which it is most distinctly heard, viz. that part of the chest wall already described to you as the mitral area—that is the area of a circle of about one inch in diameter described round the point where the heart's apex impinges as a centre. This murmur is not much propagated in any direction ; an educated ear may no doubt note that modification of the first sound which always accompanies this murmur wherever the heart's sounds are to be

heard, yet it is only over a very limited area that it is audible as a true murmur, being as a rule only indistinctly to be heard above the third rib, and its propagation is almost equally limited in every other direction. To give you an idea of the limitation of this propagation, I may mention that there is a lad of eighteen now in Ward V. with a murmur of this character. It is so loud and rough that on admission it was easily recognised through three shirts (two of them flannel), a waistcoat, coat, and topcoat. Yet when the lad is stripped this rough murmur is not distinctly audible above the third rib, nor below the middle of the sixth interspace; nor does it pass farther to the left in the nipple line than a line let fall perpendicularly from the anterior border of the axillary space; while on passing to the right the murmur is already less rough at the left edge of the sternum, and is quite lost half an inch beyond its right edge.<sup>1</sup> I give you this as excellent illustration of the remarkable limitation of propagation even of an exceedingly loud and rough presystolic murmur; but this murmur is not always limited to so small an area, though exceptions are much rarer in regard to its propagation than its character. In accordance, therefore, with the laws of the propagation of murmurs already laid down, this position of audition, as we may term it, stamps this murmur as of mitral origin, and, coupled with other concomitant phenomena, even hints its probable rhythm to an intelligent mind.

The rhythm of a murmur, as you are already aware, is its relation to the several physiological acts which constitute a complete cardiac cycle—that is those several acts which occupy the time between two consecutive apex beats. During this period we have the ventricular systole, synchronous with the apex beat, the first sound, and the first silence; the ventricular diastole, synchronous with the second sound and the second silence; and lastly the systole of the auricles,

<sup>1</sup> W. C., Ward V.—The murmur in this case passed farther to the right than usual. He subsequently died, and his case will be found detailed farther on, where the reason for this will appear; *vide* Case IX., p. 122.



which coincides with the latter part of the ventricular diastole, and immediately precedes the ventricular systole, into which, indeed, it runs.<sup>1</sup> In timing any murmur, therefore, we first of all ascertain whether it takes the place of either the first or second sound, or, if it does not, then we must determine accurately the exact relation of the murmur to these sounds, which of them it precedes, or which of them it follows, and at what interval. Now in this matter there is this fallacy, that the first and second sounds are not always audible together at either apex or base, either in health or disease, and when this is the case in disease the murmur present is apt to be taken as replacing the absent sound. This is specially apt to happen in connection with the murmur of which I am now speaking, for in the mitral region this murmur is not infrequently followed by a single loud accentuated sound, and most students at once assume the case to be one of mitral regurgitation (systolic murmur) with an accentuated second sound. To correct this error it is only necessary to time both the murmur and the sound by the pulse in the carotid artery, when the murmur will be found immediately to precede and to run up to the carotid pulse, with which the sound is distinctly synchronous. But we know that the carotid pulse is synchronous with the first sound of the heart, and precedes the second one. The sound we hear is therefore the first sound of the heart, and as the murmur immediately precedes it and runs up to it, it must occupy the time of the auricular systole. Inasmuch as the ventricular systole is the first portion of the heart's action that gives rise to audible or tangible phenomena, it is usually called *par excellence* the systole, and this murmur occurring just before it has been termed presystolic. But this is a misnomer; this murmur is truly systolic both in rhythm and character, but the systole on which it depends is that of the auricles, and not that of the ventricles; it is an auricular-systolic murmur. In timing this murmur we must be

<sup>1</sup> *Vide* diagram, Fig. 4, p. 39.



careful to employ the carotid and not the radial pulse; the former is always synchronous with the ventricular systole and the apex beat, but the latter even in health is always delayed to an appreciable extent—one-sixth of a second; while in disease, especially such as interferes with arterial contractility, this delay is notably increased, and sometimes amounts to an entire cardiac pulsation. A reference to the radial pulse is thus always embarrassing and may greatly mislead; but a reference to the carotid pulse is a perfectly safe guide, only liable to the misconception inseparable from the difficulty of comparing an audible with a tangible phenomenon. But this difficulty is trifling, and such as it is we can readily overcome it by practising on hearts which are either naturally slow or have had their action artificially retarded.

Various combinations of vowels and consonants have been employed to represent phonetically the healthy sounds of the heart and its morbid murmurs. No murmur lends itself more readily to this phonetic treatment than the presystolic, the symbols *rrrb* or *vōōt*, when vocalised, conveying to the ear sounds almost identical with those produced within the heart.<sup>1</sup> Indeed the very accent with which these symbols are pronounced may be made to represent the changes which are noted in different murmurs, or in the same murmur at different times. The last consonant in each symbol is coincident with the apex beat.

Furthermore, sounds are only audible vibrations, and in many cases these are to be felt as well as heard. The audible

<sup>1</sup> Sounds indeed which cannot be mistaken for anything but what they actually represent. Thus Duroziez scoffs at "this famous presystolic murmur of which every one speaks; which no one understands; which Hope never heard, and Bouillaud neglects and even denies." Yet Duroziez vocalises the murmur of mitral stenosis, heard at the base as *vout-ta-ta*, and heard over the apex as *ffout-ta-ta-rou*, vocables which no one at all acquainted with the matter could pronounce without recognising that he had before him a case presenting a presystolic murmur, a reduplicated second, and a mitral diastolic murmur.—Vide *Archives Générales de Médecine*, 5ième série, tome xx., p. 390, 1862.

rattle of carriages along a street is also perceived as a tremor within the houses that line it; and if we strike a tumbler sharply so as to produce a musical note, the vibrations causing this note are readily felt by a finger placed on the edge of the glass, and the sound ceases at once when we stop these vibrations. Nay, more, the sounds produced by friction of a moistened finger along the edge of glasses partially filled with water are not only to be felt as vibrations, but may be seen as crispations on the surface of the water. In like manner the vibrations which give rise to cardiac murmurs may be frequently felt by the finger at their point of origin. As the rougher the murmur is the more readily the vibrations causing it are to be perceived, and as few murmurs, if any, are so rough as the presystolic, so it more frequently than any other gives rise to a distinct sensation of vibration. This feeling of thrill is termed the *frémissement cataire*, or purring tremor; it is rarely absent in cases of mitral stenosis, being sometimes to be felt in the mitral area when the murmur, of which it is the tactile representative, is either entirely absent or only imperfectly developed. It is interesting to note in such cases that this thrill runs up to and terminates in the apex beat, just as the murmur which it represents is heard to end in the accentuated first sound.

A murmur, then, presenting the characteristics just described, which is rough and capable of being vocalised by the sounds symbolised by the letters *rrrb* or *vōōt*; which is separated from the second sound by a longer or shorter but always readily appreciable interval; which distinctly precedes the apex beat and the carotid pulse, usually running quite up to them, but occasionally separated from them by an extremely short but appreciable interval; whose rhythm may be thus graphically rendered (Fig. 11)<sup>1</sup> and which is (almost) always accompanied by a purring tremor over the mitral area; is

<sup>1</sup> Figure 11 represents graphically various phases of the presystolic murmur: (1) represents an ordinary presystolic murmur running up to the first sound; (2) represents a murmur prolonged during the whole of the ventricular diastole to the commencement of the ventricular systole; (3) represents a diastolic

invariably an evidence of deformity of the mitral valve accompanied by stenosis of the mitral opening, and is that to which alone is applicable the term presystolic, or more accurately auricular-systolic. If this murmur is once heard and recognised, then we are certain that after death we shall find more or less constriction of the auriculo-ventricular opening, and more or less deformity of the mitral valve. The murmur may, and often does disappear, but the lesion is permanent.

The way in which this murmur sometimes disappears temporarily or permanently is somewhat remarkable, and has given rise to many unlucky *contretemps* not altogether creditable to practitioners of medicine. For instance :—

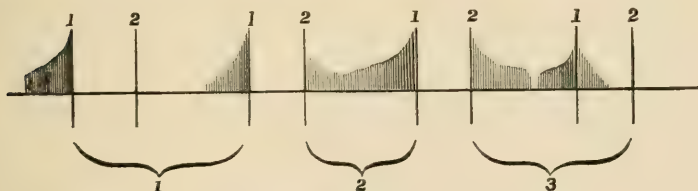


FIG. 11.

CASE VII. I well remember a gentleman with a disappearing presystolic murmur who possessed quite a bundle of certificates from medical men; one half of these testified that he laboured under organic disease of the heart, while the other half certified equally strongly that he was altogether free from cardiac disease. After a time this patient died, and his mitral valve was found to be deformed, the mitral opening constricted, and the appendix of the left auricle filled with an organised clot.

In every case of cardiac disease there are always various subsidiary phenomena which prove the existence of a cardiac lesion apart altogether from the presence of a murmur; of these I shall speak more at large presently.

murmur separated from a succeeding presystolic murmur by a short but definite pause, while the auricular-systolic murmur is seen to pass directly into a ventricular-systolic murmur.

For quite ten years after the publication of Gairdner's masterly exposition this presystolic murmur continued to be regarded, especially south of the Tweed, as both rare and difficult of detection. And it was not till after the publication (in 1871) of Hilton Fagge's comprehensive treatise on the direct mitral murmur, both from a historical and clinical point of view,<sup>1</sup> that the auricular-systolic murmur may be said to have obtained general acceptance as a special murmur perfectly distinctive of stenosis of the mitral opening. But though the cause of the murmur has thus been universally conceded, its rhythm as ordinarily understood has not been accepted by all. Thus the late Dr. Andrew Barclay of London,<sup>2</sup> Dr. Leaming of New York,<sup>3</sup> Dr. Frank Donaldson of Maryland University, U.S.A.,<sup>4</sup> Dr. W. H. Dickinson,<sup>5</sup> and others have argued that its rhythm is not auricular-systolic but ventricular-systolic, that is that the murmur does not coincide with the systole of the auricle but with the first part of the systole of the ventricle. It does indeed seem remarkable and even mysterious that the comparatively feeble and brief contraction of the auricle should be associated with a murmur so loud and rough, nevertheless the facts in favour of the ordinarily accepted rhythm of this murmur seem quite incontrovertible. These facts may be comprehensively stated as follows :—

1. There is never any doubt as to the position of maximum intensity of this murmur: this is unmistakably in the mitral area. Neither thrill nor murmur are distinctly perceptible as such above the third rib (*vide antea*, p. 113). Notwithstanding its distinct and rough character this murmur has an extremely well-defined area of diffusion, and is never propagated over the cardiac area, into the aortic area, or into

<sup>1</sup> *Guy's Hospital Reports*, vol. xvi. p. 326, 1871.

<sup>2</sup> *Lancet* (March 1872), pp. 233, 353, and 384.

<sup>3</sup> *New York Journal of Medicine*, June 1868.

<sup>4</sup> Paper from Author, read before the Medical and Chirurgical Faculty of Maryland, April 1874.

<sup>5</sup> *Lancet* (1887), vol. ii. pp. 650, 695, and 985; also *Lancet* (1889), vol. ii. pp. 779, 1032, and 1033.



the auricular area, as the soft blowing murmur of mitral regurgitation is.

2. In many cases of mitral stenosis the appendix of the left auricle is greatly dilated and lies close to the anterior chest wall in the second interspace. When this is the case the contraction of the auricle may be felt to coincide with the auricular-systolic murmur, just as in the same or other cases the contraction of the ventricle may be felt to coincide with the apex beat and the first sound, or with the systolic murmur if present.<sup>1</sup>

3. In commencing dilatation of the heart it not infrequently happens that a true ventricular-systolic murmur is found to occupy the first portion of the ventricular systole, and is followed by a first sound: precisely as some imagine the conditions to be in mitral stenosis. But a true ventricular-systolic murmur of this character is always soft and blowing, and is only heard as a murmur in the second interspace just outside the pulmonary area, where, as Walshe says, "the appendix is visible anteriorly, as the organs lie *in situ*."<sup>2</sup> In mitral stenosis the appendix of the left auricle is almost invariably dilated and in close proximity to the anterior chest wall,<sup>3</sup> yet notwithstanding the presence of conditions so favourable to the propagation of a systolic murmur into the auricular area the rough presystolic murmur is never to be heard there.

4. The direct mitral murmur, like the direct (systolic) aortic murmur, is always rough, partly, no doubt, because both are caused by the direct (onward) current of the blood, while in both cases a regurgitant murmur is soft and blowing. Between the two murmurs (systolic and diastolic) there is interposed a scarcely appreciable soundless pause, or, what is more common in the mitral area, a well-marked sound. Over

<sup>1</sup> As pointed out by Sansom, vide *Lancet* (15th Oct. 1887), p. 782; and by myself, vide p. 155; 2nd edition, p. 154; 1st edition, p. 144.

<sup>2</sup> *Diseases of the Heart*, etc., 3rd edition (London, 1862), p. 4.

<sup>3</sup> *Journal of Pathology and Bacteriology* (Edinburgh and London, 1896) p. 75; and *The Senile Heart* (A. and C. Black, London, 1894), p. 55.



the apex of a heart with a stenosed mitral opening and this combination of murmurs the sounds may be phonetically represented by the symbol *rrrbfff*. In this symbol the *rrr* represents the presystolic portion of the murmur. The *b* represents the accentuated first sound, but it may also be taken as indicating, though not as phonetically representing, that abrupt change in the character of the murmur, that interval between the two murmurs sometimes present, and which, as Gairdner has said, is almost "too brief to appear soundless to the listening ear,"<sup>1</sup> while the *fff* or *pho*, as it may also be represented, marks the soft ventricular-systolic (regurgitant) murmur.

These facts show, that whatever mystery may attend the production of so rough a murmur by the comparatively feeble and brief contraction of the auricle, all the available evidence is strongly in favour of its auricular-systolic rhythm.<sup>2</sup>

Now is perhaps the most appropriate time to refer to the opinion of the late Dr. Austin Flint, who, while agreeing as to the auricular-systolic rhythm of the presystolic murmur, yet conceived that in some three instances he had heard this murmur where, after death, the mitral valve was found to be perfectly healthy, the only discoverable lesion being free regurgitation through the aortic valve. Flint's idea was that the free regurgitation from the aorta floated the segments of the mitral valve into such close apposition that the auricular contraction, forcing the blood between these segments, produced a presystolic murmur of what he called "a blubbering character." Since Flint's day some dozen of similar cases have been placed upon record, none of them more convincing

<sup>1</sup> *Lancet* (Oct. 1887), p. 782.

<sup>2</sup> Other evidence of a similar character will be found detailed by myself at p. 714 of the *Lancet* for 25th May 1872; and by Gairdner at p. 781 of the *Lancet* for 15th Oct. 1887; also at p. 884 of the *Lancet* for 29th Oct. 1887, etc. The evidence of the cardiograph is also distinctly in favour of the auricular-systolic rhythm of this murmur; for this I may refer to Dr. Mahomed's paper in the *Medical Times and Gazette*, April 1872; and to Dr. Galabin in *Guy's Hospital Reports*, 1875; also to a paper by Dr. W. Fenwick and Mr. Walker Overend in the *Lancet* for 26th Oct. 1889, p. 843.

than his own.<sup>1</sup> In regard to this matter I can only say that I have never heard a presystolic murmur without finding after death an actual—not merely virtual—stenosis of the mitral opening; and also that free aortic regurgitation is so common an occurrence that if it could ever give rise to an auricular-systolic murmur these murmurs would be of even greater frequency than they now are.<sup>2</sup>

The condition of valve generally associated with the presystolic murmur is that usually termed a diaphragmatic valve, in which the two segments of the mitral valve are united and stretched like a diaphragm across the auriculo-ventricular opening. This diaphragm, as we may call it, is thickened especially at the edges of the central opening; sometimes it is almost cartilaginous; and its surface is usually smooth, though a few small and frequently calcareous vegetations are occasionally attached to the edge of the opening. The central opening varies in size from a button-hole too small to admit even the point of the little finger, up to an aperture not much less than normal. An unusually loud, rough, and persistent murmur by no means indicates that the opening is more constricted, or the valve rougher or denser than usual. Roughness of the murmur is quite independent of roughness of the valve, or of any remarkable hypertrophy of the auricle, as is very well shown in the following case:—

<sup>1</sup> A full account of this view and the cases on which it is based will be found in the following documents:—Flint, *Lectures on Diseases of the Heart*, (Philadelphia, 1870), p. 207; also *International Journal of Medical Science* (1886), p. 35. Gairdner, *Glasgow Medical Journal*, vol. xxviii. p. 226; and *International Journal of Medical Science* (1889), p. 137. Bramwell, *International Journal of Medical Science* (1888), p. 236. Guitéras, *Medical News*, (Nov. 1885), p. 533; and *Transactions of the Association of American Physicians*, vol. ii. p. 39. Osler, *Transactions of the Association of American Physicians*, vol. iii. p. 138. Maguire, *Medical Chronicle* (June 1890), p. 180.

<sup>2</sup> In the normal state of the heart “the segments of the mitral valve at the end of the ventricular diastole are so close as to be nearly in contact.” —Pettigrew, *Transactions of the Royal Society of Edinburgh*, vol. xxiii. part iii. p. 796; *vide* also Noel Paton “On the Action of the Valves of the Mammalian Heart,” *Transactions of the Royal Society of Edinburgh*, vol. xxxvii. part i. p. 187.

CASE VIII. Margaret Ross was an inmate of Ward XIII. for a whole year, suffering from general dropsy, depending on kidney disease (large white), for which she was repeatedly tapped, etc. What chiefly concerns us now is that she had over her heart's apex a persistent and well-marked presystolic murmur accompanied by thrill. At the *post-mortem* examination her kidneys were found diseased as expected; her heart was enlarged, weighing 15 oz., with milk spots on its anterior surface; the left ventricle was in a state of concentric hypertrophy, and its walls considerably thickened (this being ascribed to the kidney disease); the segments of the mitral valve were united and thickened throughout, but chiefly at their margins; the surface of the valve was perfectly smooth, and there were one or two minute vegetations on the edge of the central opening, which was so contracted as only to admit the point of the middle finger; the *cordæ tendineæ* were contracted and matted together; the aortic cusps were competent and natural; the right side of the heart was apparently natural.

This was a well-marked case in which, as we see, the diagnosis during life was fully confirmed by the appearances found after death.

CASE IX. William Craig had a presystolic murmur so loud and rough that I selected it as a standard of the area over which such a murmur could be heard. In mapping out this area I found that it extended unusually far to the right, and the thought occurred that possibly we might in this case have a stenosis of the tricuspid as well as of the mitral valve. But the idea was dismissed as in the highest degree improbable, and the wide area over which this murmur was heard was ascribed to its unusual loudness and roughness. As it happened, the result showed that in this I was mistaken, though there were no other symptoms or signs present but the wide area over which the murmur was heard which could countenance the former idea. The patient was anæmic, and there were consequently no jugular throbbings present,

and the increased transverse dulness of the heart could not, of course, be accepted as a definite proof of tricuspid stenosis. The patient died of latent pneumonia following, and apparently induced by scattered patches of pulmonary apoplexy (embolic pneumonia), a condition which had been recognised during life. On opening the thorax, the enormous size of the right auricle at once attracted attention. It measured inside eight inches and a half in circumference by two and three-quarters vertically, and contained a large clot, half an ounce of which was decolorised. The walls of this auricle were somewhat thickened, but irregularly, varying from one line to a quarter of an inch. The *musculi pectinati* were singularly well developed. On looking down on the tricuspid valve from the auricular aspect its segments were seen to be matted together, and the opening so constricted as only to admit the point of the middle finger; the endocardium in its neighbourhood was slightly thickened; a few small vegetations were attached to the free margin of the valve. The right ventricle was slightly dilated; its walls not hypertrophied; the *cordæ tendineæ* contracted; and no distinctive traces of the three segments of the valve were to be found. The left auricle was so dilated as to admit a ball two inches in diameter; its walls were not hypertrophied; its endocardium was thickened. The mitral opening from the auricular aspect scarcely admitted the tip of the little finger; the upper surface of the valve was covered by many calcareous spiculæ, and a few small vegetations beset its free margin. The left ventricle was apparently normal both as to walls and cavity. The aortic and pulmonary valves were competent and healthy. The rest of the dissection is omitted as unimportant. The boy's illness, according to his own statement, dated only a few months back. There was no history of rheumatism in either of these individuals.

Cases illustrative of this peculiar murmur and its accompanying lesion are of such common occurrence that it is quite unnecessary to multiply them here.



With a mitral valve deformed in this peculiar manner closure of the opening seems necessarily impossible, and regurgitation to a greater or less extent must be present in every case; yet a systolic murmur is frequently wanting, as in the two cases just narrated, while perhaps quite as frequently, as I shall presently point out, the systolic murmur is the only one distinctly audible, and in yet a third series of cases both murmurs are to be heard separated by an apex beat, a first sound, or an almost inappreciable soundless interval.

The pulse in this form of cardiac disease is naturally weakened in exact proportion to the amount of stenosis present, and is more or less irregular,<sup>1</sup> though not markedly so if no pyrexia co-exist. The following diagram may be

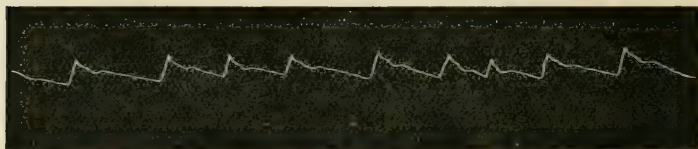


FIG. 12.

accepted as a graphic representation of an average pulse of this character (Fig. 12).

The rough presystolic murmur just described, though certainly most common, and most characteristic of diaphragmatic mitral stenosis, is far from being the only kind of murmur heard in such cases. Of the variously complicated

<sup>1</sup> Marey, in his *Physiologie Médicale de la Circulation du Sang* (Paris, 1863), p. 526, has expressed his opinion that a mitral stenosis, when sufficiently marked to give rise to a diastolic (presystolic) murmur, suppresses the irregularity of the pulse. A glance at the tracings by which he seeks to confirm this view shows at once upon what he founds it. When the stenosis is not extreme, and the compensation is good, the murmur is usually loud and rough, and the pulse tolerably full and regular. Marey has evidently taken his illustrations and based his opinions on cases of this kind. But we see such cases only incidentally. Like all other cardiac cases, those with mitral stenosis only come to us as patients when compensation fails; an early result if not also a cause of this is defective nutrition of the myocardium, and as a consequence irregularity of the heart's action and of the pulse, more marked in this form of cardiac affection than in any other, only occasionally emulated in atherosclerosis of the coronary arteries, or in cases of extreme dilatation of the ventricles, and often amounting to a veritable *delirium cordis*. Physiology is quite at one with clinical experience in this matter.



murmurs to be heard in connection with a valve thus deformed the two following cases afford striking examples:—

CASE X. Mary Macmurray, aged thirty-one, admitted into Ward XIII. on 26th May 1870, complaining of great pain across the chest, loss of appetite, and general debility. She stated that she had never been very robust, and had suffered from acute and subacute rheumatism on three separate occasions; the first of these attacks occurred nine years ago, the second seven, and the third three years ago. She also suffers almost constantly from chronic rheumatic pains in her limbs and chest. She states that her head was affected (delirious) towards the close of her first attack. About ten months ago her feebleness increased, she lost her appetite, and suffered from severe pains in the precordial region. Since that time she has got gradually worse. Her urine was found to be acid and normal in quantity; spec. grav. 1016; it contained one-fourth of albumin, and also epithelial, granular, and hyaline casts. This kidney affection was her most serious ailment, and was that indeed of which she ultimately died; but in relation to our present subject her thoracic signs and symptoms were by far the most interesting, and were as follows:—Her pulse was 104 and feeble, both radial pulses alike, and both pupils normal. The heart's apex beat behind the fifth rib, as ascertained by percussion, and two inches and a quarter to the left of the sternum. In the parasternal line one inch from the left edge of the sternum the percussion note was quite clear from the clavicle down to the upper edge of the second rib. From the upper edge of the second rib to that of the third rib the percussion sound was comparatively dull, and it was perfectly dull from the upper edge of the third rib to the lower edge of the fifth, where the tympanitic sound of the stomach became audible. Along the upper edge of the fourth rib complete dulness extended from half an inch to the right of the sternum to a distance of two and a half inches from its left edge, while for half an inch beyond this there was comparative dulness. Along the

lower edge of the second rib dulness extended for two inches and three-quarters to the left of the sternum. In the dull part between the second and third ribs on the left side a pulsation was distinctly to be felt, but less forcible than that of the cardiac apex. On listening over the apex beat a distinct presystolic murmur was usually but not always to be heard. The systolic sound was roughened and occasionally replaced by a soft blowing murmur; the second sound was followed by a loud, musical, diastolic murmur. Between the second and third ribs on the right side the first sound was heard muffled, and there was considerable accentuation of the second sound, followed by a diastolic murmur. Between the second and third ribs on the left side, close to the sternum, the second sound was more markedly accentuated, and the diastolic murmur more distinct. A little farther to the left the same sounds were heard, and a distinct sense of pulsation was conveyed to the ear by the stethoscope. The accentuation of the second sound and the diastolic murmur were, however, most distinct immediately behind the sternal end of the second rib at the left side. The accentuation was distinctly propagated into the left subclavian artery, but not the murmur. Into the left carotid both sounds were propagated, but not distinctly. Into the right carotid and subclavian both sounds were distinctly propagated. On auscultating up the right edge of the sternum, the second sound and the diastolic murmur were heard to become gradually louder as the upper edge of the sternum was approached, where it is joined by the first rib; but nowhere on the right did they attain the same loudness and distinctness as on the left of the sternum. A slight humming noise was occasionally audible in the jugular veins. For the last month of her life the patient's urine became more scanty, uræmic sickness and vomiting were frequent, and she gradually sank and died on 21st December, the immediate cause of death being an absolutely latent pleuropneumonia, which was entirely devoid of any subjective

symptoms whatever. Her cardiac signs remained unchanged to the end. At the autopsy, on 23rd December, the lower lobes of both the right and the left lungs were found hepatised, and were covered externally by a thin layer of perfectly recent lymph, presenting a honeycomb appearance. The left lung was slightly retracted, uncovering the heart to a greater degree than usual. The heart itself was purse-shaped and somewhat enlarged, its substance healthy. The aortic valves were competent, but the cusps were thickened and their whole under (ventricular) surface was covered by numerous vegetations. The mitral valve was thickened and its opening contracted, scarcely admitting two fingers, with some threads of recent lymph attached to its edge; the upper surface of the aortic segment of this valve was thickly studded with rough stumpy vegetations of varying size. The liver was healthy. The spleen weighed 15 oz., and on its posterior border had seven hæmorrhagic infarctions of a triangular shape, varying from the size of a pea to that of a small bean (of embolic origin); it was otherwise healthy. The kidneys were slightly enlarged; the right weighed 5, and the left 6 oz. The cortical substance was lessened; the capsule natural, and when peeled off exposed a smooth organ. The intestines were congested, and the rectum and lower part of the sigmoid flexure of the colon were thickened.

The next case is equally instructive, but wants the crucial decision of a *post-mortem* examination. I shall confine myself solely to the cardiac phenomena.

CASE XI. J. H., aged eighteen, was sent up to Ward XIII. from the fever house, convalescent from typhus, on 6th June 1871. She stated that two years ago she had a severe attack of rheumatism, which affected all her joints, but not her chest, and lasted for four months. After her recovery she began to suffer from pains in her cardiac region, palpitation of her heart, and shortness of breath on going upstairs. Three months previous to the attack of typhus, from which she has just recovered, she first observed some slight swelling of her

feet. Since her fever the pains in her chest have been more severe, but she has no dyspnœa while lying still in bed. Her pulse is 84, rather small, steady and regular; there is a perceptible thrill over the apex of the heart. The vertical dulness of the heart is normal; its transverse dulness in the line of the fourth rib extends from the right edge of the sternum towards the left for a distance of three inches and a half. The apex beat is between the fifth and sixth ribs, three inches and a half from the left edge of the sternum. On auscultating over the apex a somewhat prolonged murmur is heard, the first portion of which is rough and precedes the apex beat; the second portion of the murmur follows the apex beat and is soft and blowing. Between the second and third ribs on the right side there is a systolic murmur prolonged upwards and followed by a normal but feeble second sound. Between the second and third ribs on the left side, close to the sternum, there is a systolic murmur followed by a markedly accentuated second sound, which is immediately succeeded by a soft, blowing, diastolic murmur. This diastolic murmur ceases to be audible at a distance two inches and one-third from the left edge of the sternum along the second interspace. On auscultating downwards from the third rib at one inch from the sternum this diastolic murmur continues to be heard till about the middle of the fourth rib; after this it ceases, the presystolic and systolic murmurs alone being audible beneath this point. Over the sternum this diastolic murmur is only to be heard between the second interspace and the fourth rib. The systolic murmur is slightly propagated into the carotid arteries, as is also the aortic second sound which remains pure. During the progress of the case the ventricular-systolic murmur over the apex was frequently absent, and the diastolic murmur at the base became more limited in its area. This patient was discharged improved on 12th August.<sup>1</sup>

<sup>1</sup> J. H. gradually developed aortic incompetence, with all its usual results. She married in 1879, had her first child early in 1880, and died under my care from asystole due to advanced aortic disease in March 1880. I was unable to procure an examination of her body.



The murmurs in these two interesting cases differ considerably in some respects, yet both belong to the same category, examples of which, though not of everyday occurrence, can yet scarcely be called rare. The first remark I shall make is that the systolic basic murmur on the right or aortic side of the sternum, in the second case, probably depended on a similar roughened condition of the under side of the aortic cusps which we had in Macmurray. In Macmurray's case the first sound was only muffled; there was no murmur, its absence being probably due to a feeble blood-current. On the other hand, if we look upon this systolic murmur as propagated from the left side, it belongs to quite a different category, but in that case it would not likely have been so distinctly propagated into both carotids. The first point having a distinct bearing on the subject in hand—the diagnosis of mitral stenosis—is the occurrence in both of these cases of a diastolic murmur, loudest at the left edge of the sternum at the base of the heart. In H.'s case there never was any doubt as to the diastolic character of the soft blowing murmur; in Macmurray's case her ordinary pulse of 104 had to be brought down to 84, by the recumbent posture and the use of digitalis, before I could positively satisfy myself that her musical murmur was truly diastolic in rhythm. The region of the pulmonary artery, in the neighbourhood of which these murmurs are frequently heard,<sup>1</sup> has been not inaptly termed the region of romance, because of the variety of murmurs audible there which have given rise to much speculation. Laennec, it is well known, taught that the second sound was due to contraction of the auricles. Hope, though well aware that the second sound was caused

<sup>1</sup> The position of maximum intensity of this mitral diastolic murmur varies in each case; it is usually most distinct at the sternal end of the fourth rib on the left side, though sometimes it is heard in the mitral area, and frequently, as stated in the text, in the pulmonary area. The soft, slightly *musical*, diastolic murmur audible in such cases in the mitral, or more faintly in the pulmonary area, is undoubtedly mitral in character and may disappear as the disease progresses. A soft, *blowing*, diastolic murmur, heard at or below the level of the aortic valve, is just as certainly due to commencing regurgitation through this valve, and gradually increases as the disease progresses.



by the closure of the semi-lunar valves, yet taught that "when the mitral valve is contracted, a murmur accompanies, and sometimes entirely supersedes, the second sound," the influence of Laennec's teaching marring no doubt the excellence of his own observation, for a diastolic murmur of mitral origin never obscures the second sound, though it often, as in the cases just described, immediately follows it. Sometimes, as happened in them, it is separated from the true presystolic murmur by an appreciable interval [*vide* Fig. 11, p. 117, (3)]; at other times this murmur begins with the diastole and runs right up to the apex beat, thus terminating with the rough presystolic murmur [as represented in Fig. 11, (2)]. In both classes the first portion of the murmur is distinctly soft and diastolic in character and rhythm, the latter portion—whether separated from the first by a soundless interval or not—is always rough in character and auricular-systolic in rhythm; the direction of the blood-current is the same during both periods, but during the latter period we have the influence of muscular propulsion superadded and revealed by the roughening of the murmur. In Macmurray's case there is positive proof that the pulmonary valve was competent and that its condition had nothing to do with the production of the diastolic murmur. In J. H.'s case the absence of any symptoms of disease of the right heart, the rarity of affections of the pulmonary artery, and the comparative frequency of diastolic murmurs in cases of mitral stenosis, make it most probable that this murmur depends in this case also upon the same cause. The appreciable interval which, in both cases, existed between the diastolic and the presystolic murmurs makes it impossible that prolonged auricular contraction, encroaching on the ventricular diastole, could have been the cause of these diastolic murmurs. In the face of a continuously contracting auricle, no theory of the production of these murmurs could account for a soundless interval interposed between a soft and a rough murmur, though the gradual roughening of the murmur towards its close is easily explic-

able. On the contrary, if we suppose two different causes, following each other, and acting in the same direction in the production of these murmurs, we have a sufficient explanation of the difference in character of the first portion of the murmur from the second. If we further suppose the first cause to diminish to zero before the second begins to act, then we have an efficient explanation of the pause occasionally interposed between the soft and the rough murmurs, and we can readily surmise an efficient reason for the occasional absence of this pause. It must be evident that the more nearly the mechanism of the heart approaches the normal, so much the more closely will the various acts approach the physiological norm. The presence or absence of an appreciable pause in the sequence of these acts may thus be regarded as a measure of the abnormality existing, an indication of the magnitude of the obstacle to the onward flow of the blood—of the degree of stenosis present; just as the presence or absence of a diastolic murmur may also be accepted as a probable indication of an unusually great intra-pulmonic blood-pressure or the reverse, as this cannot but be a most important element in its production, seeing that no murmur can arise unless the blood passes through the constricted opening *avec une force suffisante*.<sup>1</sup>

If we take an india-rubber bag-syringe, and, after emptying it of air, insert its nozzle into water, it will be found to fill with a rapidity proportionate to the size of the opening in its nozzle. If any particle of dirt gets into this nozzle the bag either fills more slowly, or, if the opening be quite stopped, it ceases to fill at all. So it is with the heart; in the normal condition of the auriculo-ventricular valves the diastole is completed at once, the blood from the distended auricle closely following the expansion of the ventricular walls, for of course there is neither in the heart nor anywhere throughout the circulation any vacuous cavity or air-filled space into which the blood can be, strictly speaking, said to flow.

<sup>1</sup> *Vide antea*, p. 46.

Throughout the diastole the blood continues to pass into the heart, floating up the segments of the mitral valve into apposition, till the rhythmic wave of systole seizes upon the auricles, forces them into contraction, and expels their contents into the already full ventricles. When the auriculo-ventricular opening is narrowed from any cause, the ventricles—like the india-rubber bag—must fill more slowly. This delay in the expansion and filling of the ventricle must increase in proportion to the amount of stenosis present, until it may be that the wave of systole seizes the auricle before the ventricle has become filled, before its diastolic expansion is completed. In these circumstances pulmonary congestion must, of course, be very considerable, and the consequent rise of blood-pressure within the pulmonary circuit favours that reduplication of the second sound, already described,<sup>1</sup> which is so common an occurrence in mitral stenosis as to be almost pathognomonic, and which is always a matter of some importance in the diagnosis of obscure cases. The flow of blood through the contracted mitral opening in such cases of prolonged diastole may be altogether soundless, or it may be accompanied by a murmur which is probably always faintly musical, and which presents none of the harshness of the auricular-systolic murmur in which it not infrequently terminates, and this notwithstanding that both are equally direct murmurs so far as the blood-current is concerned. It seems probable that the distinctly musical character of this murmur in Macmurray's case was due to the vibrations of the stumpy vegetations covering the upper surface of the aortic segment of her mitral valve. In other cases a distinctly humming, though less musical, character may be given to the murmur by the peculiar tension of the valve itself. In the normal state of the heart a perfectly soundless interval intervenes between the close of the second sound and the commencement of the first, during this soundless period the ventricle completes its diastole and the auricle its systole

<sup>1</sup> *Vide* p. 32.

(*vide* Fig. 4, p. 39), so the more nearly the diseased heart approaches the normal one—that is the less stenosis there is—the more likely we are to have a soundless interval between the diastolic and the auricular-systolic murmurs. The absence of such an interval may, according to this view, be taken as an indication of considerable stenosis; and you will observe that Macmurray's heart bears out this view. In her case the pause was well marked, and the mitral opening actually admitted two fingers, indicating but a moderate degree of stenosis. You will observe that this explanation of the manner in which a prolonged murmur, occupying the whole of the ventricular diastole, is produced is not only consistent with all we know of the physiological action of the heart, but is also in perfect agreement with the character of the sounds heard. It also does away with all the difficulties in the way of explaining how or when an auricle is to be filled if a murmur occupying, as it frequently does, the whole of the diastole is produced by continuous auricular contraction; <sup>1</sup> the pulmonary congestion very efficiently accounting for the reduplicated second sound.

Murmurs such as these are graphically represented by the second and third illustrations [(2) and (3)], Fig. 11, p. 117, the first exhibiting these murmurs as continuous, and the second when—as in the two cases just related—there is a pause between them, a break in the shading to the right of the centre indicating this pause. Phonetically they may be vocalised by the symbols *rrrrb*, or, when softer and more musical, by *roo-oo-oo-ooob*, and when a reduplicated second is present by *rooo-oo-oo-ooob-ta-ta*. A systolic murmur in the pulmonary region or in that neighbourhood is by no means distinctive of mitral stenosis. I shall describe its mechanism and significance on another occasion.<sup>2</sup>

<sup>1</sup> At p. 709 of the *Ed. Med. Journal* for February 1871 I have gone very fully into the murmurs and other signs in Macmurray's case in connection with the diagnosis of substernal aneurysm. The occasional absence of the presystolic portion of the murmur in her case gives it a striking resemblance to that of Harriet H. (Case LXVII.) in Dr. Hilton Fagge's interesting paper on the "Murmurs attendant on Mitral Contraction," at p. 326 of *Guy's Hospital Reports*, vol. xvi. 1871.

<sup>2</sup> *Vide postea* Lecture VI.



Stenosis of the mitral opening is not always associated with a diaphragmatic valve; sometimes the valve is funnel-shaped, the opening being at the apex of the cone which dips into the ventricle. I have no dissection of any such case to relate, consequently I know not whether this form of valve gives rise to any modification of the murmurs. Now and then we have a prolonged murmur with an exaggerated first sound, which may be thus vocalised *rooo-oo-oo-lup*; this is the kind of murmur most apt to be mistaken for a ventricular-systolic murmur and an accentuated second; it is also a form of murmur that for a time I was inclined to attribute to this funnel-shaped valve. The perfect closure of the valve indicated by the *lup* in the symbol just given, seemed to be easily explained by the readiness with which the sides of a long and somewhat loose valve, with only a small opening, could be collapsed upon each other. One of the best marked of such murmurs, long under observation, at last developed a short, rough, presystolic murmur, which has since then varied frequently in character. The complete change in this man's murmur clearly indicates that as yet we have no reliable pathognomonic sign for a funnel-shaped valve.

Such then are the murmurs specially distinctive of stenosis of the mitral opening; I shall presently take up the physical signs diagnostic of this condition when there is either no murmur present at all, or when the only murmur to be heard is a systolic one. The mere addition of an apex systolic murmur to those described as distinctive of mitral stenosis in no ways affects the diagnosis; the consideration of this combination is therefore omitted.



## LECTURE V

### ON THE MURMURS AND OTHER PHYSICAL SIGNS DISTINCTIVE OF MITRAL STENOSIS, CONTINUED, INCLUDING THOSE OF INCURABLE MITRAL REGURGITATION

FROM the previous lecture we have learned that the essence of the cardiac affection in mitral stenosis is obstruction to the onward current of the blood at the auriculo-ventricular opening from deformity of the mitral valve. We deduce this from the fact that the only murmurs distinctive of this lesion are those which, from the position in which they are best heard, are known to originate at the mitral opening, and from their rhythm are recognised as occurring during the ventricular diastole, and the most characteristic during that part of the ventricular diastole occupied by the auricular systole. These murmurs, as described, are perfectly distinctive of mitral stenosis, and once the existence of one or other of them has been satisfactorily ascertained deformity of the mitral valve may be predicted with the perfect certainty that it will be found after death. These murmurs are unfortunately not always to be heard; in most cases the diastolic portion of the murmur is wanting, and in many even the auricular-systolic portion is absent for long, if not for always, as we know from a hospital history of two and even of three years. Moreover each portion of the murmur may vanish temporarily for a longer or shorter period, and this sometimes in the most puzzling and unaccountable manner.<sup>1</sup> Further, as a

<sup>1</sup> In the *Practitioner* for December 1873, p. 481, Dr. Gowers has directed attention to the influence of posture in altering the character of these murmurs, and has pointed out that in some the presystolic murmur is absent while the

necessary result of the condition of the valve in the most usual form of mitral stenosis (the diaphragmatic valve), we have a permanent regurgitation into the auricle revealed by a variety of murmurs. Sometimes a systolic murmur in the auricular area is all that is to be heard ;<sup>1</sup> in the larger number of cases in which the presystolic murmur is present, this regurgitation is not accompanied by any ventricular-systolic murmur in the mitral area. In a smaller number of cases both the auricular-systolic and the ventricular-systolic murmurs are constantly present in the mitral area. In a much smaller number these murmurs present themselves in a most irregular fashion, both murmurs being audible for one or two beats, and then the auricular-systolic or the ventricular-systolic—chiefly the latter—recurring by itself for an irregular number of beats, while in a considerable number of cases, even when very great stenosis exists, the ventricular-systolic murmur alone is to be heard and is always present. Moreover, when the patient gets enfeebled from any cause, a ventricular-systolic murmur often replaces the pre-existing auricular-systolic murmur, and this may again return should the patient regain his strength.

These, then, are the various and often complicated varieties of murmur to be heard in cases of mitral stenosis. It would be easy to narrate cases illustrative of each variety, but it is quite unnecessary, and would only oppress your memory with needless details. So far as murmurs are concerned, those only are distinctive of mitral stenosis which have been already described, and of these the most characteristic is the presystolic ; whenever that is recognised as persistent, or can even be picked out as occasionally recurring amid a complication of other murmurs, the case is quite clear and needs no further proof. When, however, no presystolic murmur, or possibly no murmur at all is to be heard, then we are forced patient is erect, and is only to be heard when he is recumbent. This only holds good for a minority of such cases ; in the greater number the presystolic murmur is most distinct, and often only to be heard in the erect posture, and after exercise.

<sup>1</sup> *Vide* Lecture VI.

to unravel the case by a careful investigation of the various subsidiary phenomena evolved during cardiac action, as well as of the relative condition of the different cardiac cavities. In this way we shall frequently be able to detect a stenosis of the mitral opening when the only murmur audible is one of regurgitation, or even when no murmur at all is to be heard.

Amongst the most remarkable of these subsidiary phenomena must be reckoned *irregularity of rhythm*, which is almost invariably present in a greater or less degree,<sup>1</sup> and is sometimes so marked as to be a diagnostic phenomenon of considerable importance. This great irregularity appears to be due to defective nutrition of the myocardium and its ganglia. It is not in these cases due to dilatation of the left ventricle, because in mitral stenosis that is comparatively rare and is never excessive. This irregularity is often just as little marked in cases of great dilatation of the right ventricle, as when the left ventricle is hypertrophied, and the right ventricle comparatively unaffected. On the other hand general debility seems to have considerable influence in developing this irregularity, and it is always lessened by measures directed towards improving the general health and especially the cardiac power. But it is chiefly the co-existence of pyrexia which most strikingly conduces to the development of this irregularity, and that quite independent of the degree of stenosis present. Indeed irregularity is prone to occur in other forms of cardiac disease whenever pyrexia is developed, affording thus a proof that it is due to exhaustion of the energy of the myocardium and its ganglia.<sup>2</sup>

<sup>1</sup> Naturally I speak here only of those with ruptured compensation, not of those in whom the lesion is dynamically compensated, nor of those in whom compensation has been restored by treatment, *vide* note p. 124.

<sup>2</sup> The reserve energy of the heart (*vide* p. 84), upon which its recuperative power when exposed to injury or disease depends, seems to result from the fact that the heart works well within its powers, and is able to exercise its function for hours (Panum) or for many minutes (Von Bezold) after it has been completely deprived of arterial blood. Long continuance of imperfect nutrition materially impairs the cardiac metabolism and necessitates anabolic

In mitral stenosis the cardiac nutrition is always below par; in this state, therefore, this organ is specially obnoxious to lesions of innervation affecting motility. The following diagram graphically represents this extreme irregularity in a sufficiently marked manner (Fig. 13).

CASE XII. William Donald, aged thirty-seven, admitted to Ward V. on 10th March 1871, was a labourer, and had been recently working in a gaswork, where he had been much exposed to sudden alternations of heat and cold. He had always enjoyed good health till four weeks ago, when he caught a severe cold; since then he had been feverish and ill, with increasing debility, some cough, and bloody expectoration.

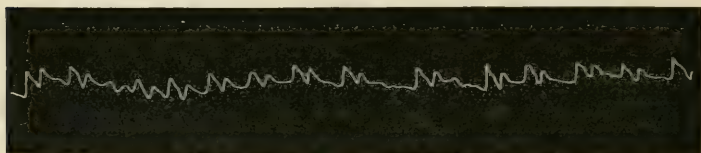


FIG. 13.

On admission he looked exhausted, his breathing was hurried, his pulse 120 and extremely irregular. On percussion the cardiac dulness was found to be normal, the apex beat in its normal position. On auscultation in the mitral area the first sound was found to be slightly thumping, impure, but unaccompanied by any murmur; no thrill was perceptible over the apex. Between the second and third ribs on the left side close to the sternum, the second sound of the pulmonary artery was heard greatly accentuated. Between the second and third ribs on the right side close to the sternum, the aortic second was comparatively weakened. At the base generally, but most distinctly at midsternum on a interference whenever the heart is called upon for greater exertion than it is equal to. The interference between anabolic and katabolic action thus brought about in the interest of self-preservation sets up irregular action (Roy and Adami, *Transactions of the Royal Society*, vol. clxxxiii. p. 293, etc.), and previous malnutrition has a most important influence in necessitating this anabolic interference. Hence the obstinate irregularity of advanced mitral stenosis, of atherosclerosis of the coronaries, and even of a greatly dilated heart.



level with the fourth rib, marked reduplication of the second sound was heard. On percussion over the left lung anteriorly, the upper lobe was normal, and the lower lobe somewhat dull; posteriorly on the same side there was a small dull patch about the centre of the scapular space; from the lower border of the scapular space downwards the lung was dull. Over the right lung the percussion note was everywhere normal except at the lower border of the scapular space, where it was dull. Over the dull portions the respiration was more or less obscured; over the rest of the chest it was vesicular, mingled with occasional coarse crepitating rattles. The expectoration was catarrhal, largely mixed with pure blood.

The diagnosis was bronchial catarrh, occurring in a patient affected with mitral stenosis, and complicated with pulmonary apoplexy (embolism with infarction).

The history of an ordinary febrile attack, accompanied with cough, catarrhal expectoration, and occasional coarse crepitation over the chest, persisting for four weeks after seizure, sufficiently confirmed the first part of this diagnosis. The copious expectoration of blood, coupled with the presence of dull patches, over which the respiration was obscured,<sup>1</sup> also confirmed the latter part of it. While the diagnosis of mitral stenosis was based upon—1st, the slightly thumping character of the impure first sound; 2nd, the reduplication of the second sound, which, though found in other circumstances, is never so persistent as in mitral stenosis; 3rd, the weakened character of the aortic, and the markedly accentuated character of the pulmonary second sound; and, 4th, the extreme irregularity of the pulse, always an indication of weakness (imperfect metabolism) of the myocardium, and therefore confirmative of the diagnosis of mitral stenosis when other signs point in that direction.

<sup>1</sup> The bronchi passing through the hæmorrhagic masses were quite occluded, there was no air within them; no resonance could therefore be developed within them, hence there was no bronchial breathing to be heard, notwithstanding the solidification of the lung—a fact that goes to confirm Skoda's theory of the origin of this phenomenon.



The thumping character of the first sound duly recognised by a practised ear is, I believe, quite pathognomonic of mitral stenosis; it is the accentuated first sound which generally follows the rough presystolic murmur, the *b* of the vocalised apex sounds *rrrrb*—all the *r*'s being expunged. This variety of the apex sounds in such cases is associated with a feeble heart, and therefore with more or less irregularity of the pulse, though this is not often so extreme as it was in the case before us. The persistent reduplication of the second sound was strongly confirmative of the diagnosis, especially when associated with the signs just referred to, and this was still further strengthened by the subsidiary, and, from a diagnostic point of view, comparatively unimportant phenomena of pulmonary accentuation and hæmoptysis.

After admission the patient gradually fell into a state of low muttering delirium, from which he was roused by the moderate exhibition of stimulants and the free use of digitalis. For a couple of days he seemed to rally, and faint hopes of ultimate recovery were beginning to be entertained, when symptoms of subacute peritonitis set in, and two days afterwards he died, on 20th March, ten days after admission, the cardiac symptoms remaining unaltered to the close. At the dissection the small intestines were found matted together with recent lymph; they were congested and friable, and the omentum was adherent to their surface, 3 oz. of bloody serum being found in the recto-vesical pouch, etc. The heart weighed  $17\frac{1}{2}$  oz. The right auricle was distended; the left auricle flaccid. Both ventricles were distended with black clots; the right ventricle was somewhat dilated, its auriculo-ventricular opening admitted seven fingers; the left ventricle was somewhat hypertrophied, its cavity not dilated. The aortic and pulmonary valves were competent; a small vegetation was attached to the *corpus aurantii* of the posterior cusp of the aortic valve. On laying open the left auricle several vegetations were seen encroaching on the auriculo-ventricular opening, one of them quite cartilaginous but

movable, the others softer. The mitral orifice admitted two fingers easily; its aortic cusp was shortened and thickened, especially at its free margin. Each pleural sac contained about a pint of fluid. Over the right lung there were many adhesions; over the left only a few at the apex. The upper lobe of the left lung contained two dark-coloured solid hæmorrhagic masses just beneath the pleura, one two inches and a half from the apex on the posterior aspect of the lung, the other two inches and a half from the interlobular fissure on its anterior aspect; the lower lobe contained four similar masses, all just in contact with the pleura, two as large as an orange, the other two each the size of a walnut; the largest was in the interlobular fissure, and extended down to the base; the next largest was in the posterior aspect of the lung. The apex of the lower lobe of the right lung contained four hæmorrhagic masses.

These *post-mortem* appearances, extracted from the pathological records of the Royal Infirmary, thus completely confirmed the diagnosis.

In the following very similar case the diagnosis was confirmed, fortunately for the patient, not by any pathological research, but by improvement in the patient's strength and the subsequent development of those murmurs which are recognised as pathognomonic of the condition diagnosed:—

CASE XIII. A. M., aged thirty-one, admitted to Ward V. on 23rd March 1871. This patient had not previously suffered from rheumatism, nor from any serious illness, and had always been able for his work up to the commencement of his present illness, which dated only five days back. He had then been seized with shivering and slight febrile symptoms, accompanied almost from the first with hæmoptysis. He was somewhat breathless, with a hot skin, some cough, copious bloody expectoration, pulse 120, extremely irregular, its sphygmographic tracing closely resembling that of the preceding case. On examination his heart was found nearly normal in size, the apex beating between the fifth and sixth ribs slightly within the nipple line. The heart's action

was extremely irregular, and on placing the hand over the apex, a thrill running up to the apex beat was occasionally, but not always to be felt. On auscultating over the mitral area, the first sound was found to be impure and slightly thumping in character, but no murmur was to be heard. The pulmonary second was accentuated, the aortic somewhat weakened; the two sounds seemed not to be simultaneous, but presented the phenomenon of reduplication, which was most distinctly to be heard about midsternum opposite the end of the fourth rib. Auscultation and percussion were normal over both lungs, except posteriorly in the infra-scapular regions, where some dulness existed over both lungs, and the vesicular murmur was much weakened, almost extinct. The diagnosis in this case was slight febricula, complicated with mitral stenosis and pulmonary apoplexy (embolism with infarction). This patient was treated with ten minims of tincture of digitalis and of squill in water every six hours. Under this treatment the hæmoptysis gradually lessened, the pulse became more regular, and fell to 90, and after about a fortnight's treatment an ordinary presystolic murmur was heard in the mitral area, and a diastolic murmur became audible at the end of the fourth rib just where it joined the sternum. The reduplication of the second sound continued to the last. He was sent to the Convalescent Hospital, and at the end of three weeks re-admitted for a slight exacerbation of his cardiac symptoms, but without hæmoptysis. The murmurs continued as described till he was discharged relieved and declaring himself fit for work.

These cases indicate in a marked manner the importance of great irregularity of the heart and pulse as a symptom of mitral stenosis, especially when associated with hæmoptysis. Extremely suspicious under all circumstances, these symptoms may, I believe, be regarded as quite pathognomonic when associated with persistent reduplication of the second sound, especially if accompanied with a presystolic thrill, notwith-

standing the absence of all murmur. Pulmonary accentuation is of comparatively slight diagnostic importance, as it is usually absent and rarely well marked when reduplication is present, and is really due to a less degree of pulmonic congestion, which may be consequent on many various lesions.

Apart from the presence of any presystolic murmur, or of any great irregularity of the pulse, mitral stenosis is not infrequently revealed solely by a systolic mitral murmur; it is therefore always a matter of interest and sometimes of importance to determine the exact nature of the lesion upon which this murmur depends. The two following cases, besides the individual interest which they possess, are of consequence as showing some of the data upon which this diagnosis may be founded.

CASE XIV. Agnes Gunn, aged eighteen, was admitted into Ward XIII. on 27th October 1870, complaining of dyspnœa, cough and expectoration. There was no history of rheumatism, but she had suffered from chorea about two years ago. About twelve months ago she was admitted into Ward XI. under Dr. Laycock, with symptoms similar to those now present, and in the record of her case it is stated that she then had a mitral murmur succeeding and partly replacing the first sound, and that the tricuspid dulness was increased. At the date of her admission under my care, her face was observed to be full, puffy, and rather livid; her expression was dull and heavy, yet not devoid of anxiety. There was some œdema of her feet and legs, great dyspnœa, considerable hard cough, and copious watery expectoration. On laying the hand over the lower part of the sternum, the right ventricle was felt to beat with a slow, heaving pulsation; the impulse in the normal position of the apex was only faintly to be felt; that between the third and fourth ribs on the left side was greater than normal. On percussing in the parasternal line one inch from the left edge of the sternum the cardiac dulness was found to extend from the upper border of the third rib down to the liver dulness. On



the level of the fourth rib this dulness extended from one inch to the right of the sternum transversely across for a distance of six inches. On auscultation in the mitral area the apex beat was found to be accompanied with a slight thump, followed by a murmur which completely replaced the first sound. The second sound was distinctly audible at the apex. On carrying the stethoscope to the left the systolic murmur gradually faded, and about the middle of the infra-axillary space was replaced by an impure first sound which continued to be heard round to the base of the scapula. Over the tricuspid area a loud blowing murmur completely replaced the first sound. At the base between the second and third ribs on the right side an impure first sound was heard followed by a feeble second. Between

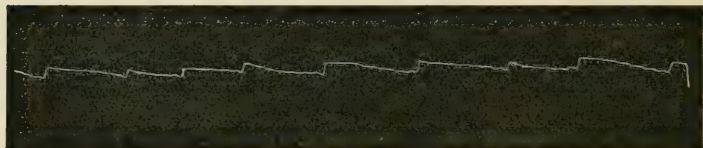


FIG. 14.

the second and third ribs on the left side the first sound was also heard impure, and was followed by a distinctly accentuated second sound. There was no venous pulsation to be seen in the neck, but the veins were small, empty and obscured by general superficial turgidity. The pulse was 76, small, feeble and irregular; its character is graphically represented in the annexed diagram (Fig. 14).

Agnes Gunn continued under treatment—mainly with digitalis and squill—till the 29th of December, when she was discharged, relieved of all her more urgent symptoms.

The elements mainly relied upon in this case, in proof of the existence of a mitral stenosis, were—1st, The slight thump preceding the systolic murmur, an imperfect and abortive attempt at a first sound frequently observed in those cases of mitral stenosis in which the presystolic murmur is



absent. It is equivalent to the *b* of the phonetic symbol formerly given, and may be looked upon, either as part of the first sound, or perhaps rather as the tail and only remaining part of the rough presystolic murmur; it is generally too sharp and short for my ear accurately to time. In other cases it is undoubtedly due to imperfect closure of the mitral, as can be accurately ascertained by timing it by the carotid pulse; in such cases it is an impure first sound, the thump is more prolonged and the systolic murmur frequently absent. 2nd, The fact that the systolic murmur was not distinctly propagated beyond the middle of the infra-axillary space. This is a common, but not an invariable occurrence in mitral stenosis; the systolic murmur is not lost but the remnant of the first sound is so much more distinctly propagated that it speedily becomes merely an impure first sound, in which the systolic blow is always more or less clearly to be recognised. This is never the case in a purely regurgitant murmur; such a murmur may become fainter as we pass to the angle of the scapula, but it never assumes any of the elements of a first sound. These two phenomena are perfectly distinctive; they are never observed save when mitral obstruction exists. As corroborative signs we had dilatation of the left auricle, shown by the unusual pulsation above the fourth rib, which was both seen and felt to precede the ventricular pulsation; and the early occurrence and rapid development of dilatation of the right auricle and ventricle, indicating considerable obstruction to the onward flow of the blood, an obstruction still further indicated by the small and feeble arterial pulse (*vide* upstroke of tracing in Fig. 14), the weakened aortic second, and the persistence of pulmonic accentuation in spite of free tricuspid regurgitation as evinced by the loud tricuspid murmur. This proof, from the state of the right heart, is even stronger in those cases in which there has been no co-existent bronchitis, as is occasionally observed.<sup>1</sup> Even

<sup>1</sup> Especially in Ellen Harkins, admitted to Ward XIII. on 20th April, and discharged improved on 12th June 1871. In her we had all the percussion

in this case, though there was evidently great pulmonary congestion and copious expectoration of watery fluid, yet the absence of rhonchi and the presence of crepitation showed that the condition present was more allied to the general turgescient œdema of the body than to true bronchitis; that we had, in fact, to do with an œdema of the lungs, rather than with a true catarrhal condition; it was therefore all the more valuable a proof of obstructed circulation.

On 27th March 1871 Agnes Gunn was readmitted for an exacerbation of all her symptoms, induced about a month previously by going from the wash-tub to an outside well one cold day while scantily clothed. Her cough, shortness of breath, watery expectoration, and cardiac distress were much increased. The jugular veins, though still small, were now seen distinctly to pulsate synchronously with the heart; her face, neck, and extremities, especially the lower limbs, were very œdematous. Her heart was much as formerly, with this exception, that the impulse just beneath the lower end of the sternum was less marked, and the apex beat was more readily to be felt. This, it was suggested, was probably due to increased dilatation of the right ventricle, which had pushed the left ventricle backwards into the chest and thrust itself into the position usually occupied by the true apex. This was evidently the only explanation possible in the face of an increasing cardiac affection, now fast becoming serious, which from the first had so largely implicated the right heart. It was impossible to suppose that a left ventricle, defectively nourished and from the first unable to hold its own, could have recovered itself sufficiently to re-assert its position, apart from any marked evidence of improvement in the onward circulation, which was entirely absent. The view taken was perfectly consistent with the usual progress in such cases; the change effected is only rarely observed in so extreme a degree. Gunn remained in Ward XIII. till her

signs of tricuspid dilatation, with distinct jugular pulsation, but no tricuspid murmur, and an entire absence of any history of bronchitis.

death, on the morning of 12th July. During this period she had an attack of hemichorea; the sufferings induced by this were rapidly subdued by hydrate of chloral and full doses of arsenic, but it persisted for many weeks as a slight twitching of the thumb and forefinger of the right hand, finally disappearing many weeks before her death. This choreic attack rapidly broke down her cardiac energy, and she never rallied, dying under symptoms of pulmonary apoplexy, with gradually increasing cardiac asthenia. The autopsy was made on 13th July, thirty hours after death. Her body was moderately fat; her legs very œdematous; her face and lips livid. On opening the thorax the pericardium was found distended by from 15 to 20 oz. of slightly turbid serum. The heart was considerably enlarged, weighing  $16\frac{1}{2}$  oz. The right ventricle concealed the left, and formed the apex of the heart; it was much dilated, and its walls hypertrophied, measuring half an inch in thickness. The tricuspid valve was slightly dilated, admitting five fingers easily; on its right cusp, near the free margin, there was a small vegetation about the size of a millet seed, soft and elastic. The right auricle was greatly dilated, not hypertrophied. The aortic valve was competent. The left ventricle was slightly hypertrophied, not dilated. The mitral opening was so contracted as barely to admit the point of the little finger; the valve itself was much thickened, and its cusps glued together by their margins; on the auricular surface of each cusp there was a row of small vegetations like millet seeds. The left auricle was slightly dilated, and its walls much hypertrophied, being about twice their natural thickness. The left lung contained two large and recent hæmorrhagic clots (infarctions) presenting on section a dark venous surface. One of these was situate along the anterior margin of the superior lobe, extending from the apex downwards for a distance of four inches, while it measured transversely one inch and a half. The other was in the inferior lobe, near its anterior margin, and was about the size of a large orange; its circumference

was pretty sharply defined. The right lung contained no recent extravasations, but in the inferior lobe, immediately beneath the middle of its external surface, there was a yellowish-gray wedge-shaped patch, with its base to the pleura, evidently the remains of an old embolic infarction. There were several emphysematous patches over the surface of the right lung, especially on its anterior margins. The liver weighed 4 lbs. 2 oz., was much congested, and slightly cirrhotic, the lobules being very distinctly differentiated by the interlobular cellular tissue. The spleen weighed 5 oz., was of firm texture, and contained two hæmorrhagic infarctions, each about the size of a walnut, tough in texture, and of a bright yellow colour. The kidneys were healthy, and weighed each  $5\frac{1}{2}$  oz. The dissection in this case also completely confirmed the diagnosis made during life. The marked hypertrophy of the left auricle, in contrast with the simply dilated condition of the right auricle, in spite of the great preponderance of regurgitation on the right side, is a sufficient indication of the influence of obstruction in developing hypertrophy as a supplementary *vis a tergo*, while it is a singular fact, from a diagnostic point of view, when we consider the entire absence of any presystolic murmur in this case.

The next case I shall quote is also instructive as to the signs upon which we have to rely for the diagnosis of mitral stenosis in those cases in which there is only the ventricular-systolic murmur of regurgitation to be heard.

CASE XV. Alexander Milne, a printer, aged seventeen, was admitted into Ward V. on 28th November 1870, complaining of palpitation, cough, shortness of breath, and pain in the cardiac region. From his history it was gathered that he had suffered from similar cardiac symptoms from the age of six, and that to these a cough had been superadded about a year ago. Since that time the pain in the cardiac region had been much worse. The patient was pale and anæmic-looking; he had never any rheumatism; his family history



was unimportant. On inspection, greater pulsation than usual was visible over the cardiac region, especially at its lower part. The apex beat was distinct between the fifth and sixth ribs, two and five-eighth inches from the left edge of the sternum. On percussing downwards in the parasternal line one inch from the left edge of the sternum, cardiac dulness was found to commence at the upper edge of the third rib, and extended down to the liver dulness; on the level of the fourth rib dulness did not commence till the right edge of the sternum, but it extended to the left for a distance of fully three inches. On auscultating over the mitral area, the first portion of a first sound was heard followed by a loud systolic murmur, closed by an imperfectly heard second sound. This systolic murmur was propagated to the right, and was distinctly heard over every part of the right ventricle, followed by a reduplicated second. It was not so distinctly propagated to the left, becoming very faint on passing the middle of the infra-axillary space. Over the right ventricle a slight but distinct heaving was communicated to the ear by the stethoscope. Between the second and third ribs on the left side, distinct accentuation of the pulmonary portion of the second sound was heard, the sound being here reduplicate. It was also reduplicate on the right side, where, between the second and third ribs, the aortic portion of the second was heard tolerably natural. Over the jugular veins a hæmic murmur was audible, but no pulsation was to be seen. The pulmonary physical signs were normal, and the cough left him entirely after a short residence in the Infirmary. The digestive system was normal, except that there was but little appetite for food. The urine was smoky, contained numerous blood-corpuscles, a few small granular casts, and one-sixth of albumin.

The peculiar character of the first sound, the mode in which the systolic murmur was propagated, and the persistent reduplication of the second sound, gave me reason to state that in this case also we had to do with a stenosis of the

mitral opening. The loud tricuspid murmur, and the evident dilatation and hypertrophy of the right ventricle, gave a seriousness to the prognosis, which was not lessened by the persistent hæmaturia and the anæmic condition of the patient.

Under treatment the patient's cough speedily ceased, his cardiac symptoms moderated, the pain left him, even the kidney symptoms were modified, the tube-casts disappeared, the hæmaturia lessened, and his general health was much improved. He was sent to the Convalescent Hospital on 23rd February. After being some time at home Milne became gradually worse, weaker, and more distressed by his cardiac symptoms, and in this state he was readmitted to Ward V. on 6th April 1871. His cardiac signs were unaltered, but his weakness was greatly increased. The hæmaturia continued, and there was a good deal of general œdema. In a short time he was confined to bed suffering much from orthopnoea, and after lingering on in this state till the 24th of May, he died exhausted, having spat a good deal of blood during the last few days of his life. At the autopsy there was found great general œdema of the body. The face, which had been livid before death, had assumed a roseate hue, and a quantity of froth exuded from the mouth. Over both lungs there were numerous pleuritic adhesions; the left lung was congested and œdematous; the inferior lobe of the right lung was hepatised, the middle lobe partially consolidated, the base of the upper lobe was also partially consolidated, and the whole lobe was œdematous. The heart weighed 22 oz. The pericardium was universally adherent; the adhesions were tough and fibrous. The right side was dilated and filled with dark clot; the tricuspid opening admitted five and a half fingers. The left side was excentrically hypertrophied and filled with a partially decolorised clot; the mitral opening was constricted, admitting only one finger and a half; its cusps were adherent and greatly thickened. The aortic valve was competent. The nutmeg

liver weighed 3 lbs. 9 oz.; the spleen weighed 12 oz.; the kidneys weighed each  $6\frac{1}{2}$  oz.; they were enlarged, the capsules non-adherent, the surface a marble-gray studded with stellate vessels and hæmorrhagic spots, the cortical portion speckled with yellowish opacities.

These cases are sufficient to show the peculiar character of that ventricular-systolic murmur that indicates its dependence on a stenosed condition of the mitral opening, and that quite apart from the presence of either of those murmurs—the auricular-systolic or the auricular-diastolic—which are known to be usually connected with this condition, both of which are frequently absent in such cases as those referred to. These cases completely disprove the idea that actual mitral regurgitation is never present unless we can carry the murmur distinctly round to the lower angle of the left scapula. But it would be equally erroneous to suppose that even when we can carry the ventricular-systolic murmur of regurgitation right round to the inferior angle of the left scapula, unpreceded by any thump and unmodified by any attempt at a first sound, we have necessarily to do with regurgitation only and not also with stenosis. Among many instances to the contrary I have selected the following, not only as a well-marked example of the condition I speak of, but also as an instructive instance of the changes which the murmur of mitral stenosis may undergo in the progress of the disease :—

CASE XVI. Andrew Ormiston, a miner, aged seventeen, admitted to Ward V. on 1st November 1869, complaining of shortness of breath and pain in the cardiac region. The patient stated that he had been ailing for about a year, but before that had never ailed, with the exception of having had chicken-pox and scarlatina in childhood; in particular he had never had rheumatism. He had been in hospital once during the past year for complaints similar to those he now had. The patient's shortness of breath was aggravated by coughing or by exertion; his expectoration was grayish, but sometimes

bloody. He had occasional paroxysms of pain in the epigastrium, coming on about half-an-hour after taking food. He was small for his years, but of a ruddy countenance and fresh, healthy complexion. He was pigeon-breasted, the chest being flattened laterally. On palpation a thrill is felt over the heart's apex preceding its impulse. Cardiac dulness extends from the upper edge of the third rib down to the liver dulness, and transversely along the upper border of the fourth rib from the right edge of the sternum to the left nipple, a distance of four and a half inches. On listening in the mitral area a well-marked rough murmur was heard to precede and run up to the first sound. The pulmonary second sound was accentuated; the aortic second somewhat weakened. The accentuation of the second sound was markedly increased whenever he happened to cough, and also after exertion. His pulse was 88, soft, and only slightly irregular. The respiratory system was normal. Tongue clean, appetite variable, bowels regular, urine normal. During his residence in hospital his general symptoms varied as he happened to suffer from incidental catarrh or dyspepsia. He was sent to the Convalescent Hospital on 15th December, and was recommended to give up mining, and if possible to procure some easy indoor occupation. For some time he took charge of a village library; latterly he assisted in a grocer's shop. He grew considerably, and became a well-grown, healthy-looking lad of his years, though quite unfit for manual labour, as his sufferings were always increased by any unusual exertion. He came occasionally under observation, without showing any material alteration in his symptoms, until 29th May 1873, when he again came under treatment with his condition in every way materially deteriorated. His history was that in the preceding February he had a severe bronchitic attack, which confined him to bed for three months, and caused serious aggravation of all his symptoms. So soon as he was able he returned to Ward V., where he was found to labour under considerable dyspnoea, aggravated by the



slightest exertion, occasional palpitation, and slight cough. His heart was found to have undergone a most serious alteration for the worse. There was distinct jugular pulsation; great and manifest heaving at the lower part of the sternum, with very evident pulsation above the fourth rib and to the left of the sternum, preceding the ventricular pulsation. At the level of the fourth rib the transverse cardiac dulness was much increased, and measured no less than six inches across, extending for two inches and a quarter to the right of mid-sternum. There was still some thrill to be felt in the mitral area, over the diffuse apex beat, the point of strongest impulse being between the fifth and sixth ribs, three inches and a quarter from mid-sternum. In this area a presystolic murmur could no longer be heard, but instead a loud systolic murmur, which was propagated distinctly round to the lower angle of the scapula, as well as less distinctly into the aortic and pulmonary areas. In the mitral area no second sound was to be heard. In the tricuspid area there was a very loud systolic murmur of a somewhat higher pitch, and a distinct second sound. In the aortic area the second sound was much weakened. In the pulmonary area the second sound was reduplicated and accentuated. Under appropriate treatment, of which digitalis was the most important part, he gradually improved, his heart contracting so that after only a few weeks' treatment the tricuspid murmur was replaced by a first sound, and the mitral murmur became preceded in the mitral area by a portion of the first sound, and in the middle of the infra-axillary space the first sound wholly replaced the systolic murmur. But the feeble constitution had received too severe a shock ever fairly to rally; the myocardium never fully regained its former tone. The slightest illness or fatigue, the mere ordinary life-work of the day, seemed sufficient to exhaust the heart and lessen its contractile force, so that the almost normal tricuspid sound heard in the morning was often replaced at night by a distinct systolic murmur. The presystolic murmur never

returned. He was sent to the Convalescent Hospital on 14th August 1873, considerably improved. Ever after he continued feeble, and frequently came under treatment for cardiac symptoms or pulmonary complications, and for the last time on 4th September 1874. His cardiac signs were much as last described. A loud systolic murmur, free from every trace of presystolic thump or of a first sound, was heard in both mitral and tricuspid areas, and was carried right round to the lower angle of the scapula, and propagated into both the aortic and pulmonary areas. These sounds continued unchanged till his death on 5th October 1874.

At the autopsy the body was found to be well nourished, there was œdema of the lower limbs, 6 to 8 oz. of fluid in the pericardium, and some effusion into both pleuræ.

The heart was much enlarged, weighing 17 oz.; all its chambers were dilated. The right ventricle was specially dilated and elongated, so that it projected beyond the left ventricle, and formed the true apex of the heart. The cavity of the left ventricle was found to be slightly dilated, and its walls very much hypertrophied. The mitral valve was extensively diseased; its opening admitted only the tip of the little finger; its cusps were greatly thickened, and adherent at their edges; each cusp contained calcareous matter. The left auricle was greatly dilated, and somewhat hypertrophied. The aorta was below its natural size, its lumen being only half an inch in diameter. The aortic valve was competent and natural. The right ventricle was greatly dilated and hypertrophied. The pulmonary artery was also greatly dilated, its lumen measuring one inch in diameter. The valve was competent. The tricuspid orifice was dilated, admitting seven fingers. The right auricle was greatly dilated, but not hypertrophied.

The right lung weighed 1 lb. 15½ oz., and contained several circumscribed masses of effused blood, some quite dark and recent, others pale and gray. The whole of the middle lobe was thus solidified. The lung was free from pleuritic

adhesions. The left lung was bound to the chest wall by firm adhesions. It was engorged and cedematous. The base was solidified as if from some old pulmonary infarction. The liver weighed 3 lbs. 10½ oz. It was intensely congested with venous blood, somewhat cirrhotic, and also somewhat fatty. The spleen weighed 6 oz., and was hard, fibrous, and congested. The kidneys weighed 5½ oz. each, and were somewhat congested.

It would not be easy to find a more instructive case than this, where we had five years of intelligent observation of a most interesting cardiac lesion, followed by a careful *post-mortem* examination. This case is indeed an epitome of all that is most interesting in the history of mitral stenosis, with the sole and somewhat remarkable exception that though the murmurs varied they did not vary capriciously, nor were they at any time much altered by the position of the patient. So long as any trace of a presystolic thump remained it was most perceptible in the upright posture, and was much increased by exercise, but when this presystolic thump had finally disappeared it was not restored either by position or exercise, and the case then presented a well-marked example of incurable mitral regurgitation—that is, a regurgitation depending upon actual disease of the mitral valve, in contradistinction to curable mitral regurgitation depending on loss of tone of the myocardium, of which I shall have more to say presently. In all stages of this affection well-marked pulsation occupied the position of the dilated left auricle above the fourth rib to the left of the sternum, and this pulsation could be both seen and felt to precede that pulsation below the fourth rib, which coincided with the apex beat. The superior pulsation, both in position and rhythm, was coincident with the left auricle and its systole, just as the inferior pulsation was coincident with the left ventricle and its systole. The auricular pulsation was, as it always is, synchronous with the thrill running up to the apex beat and with the presystolic murmur, showing clearly the dependence

of both of these phenomena upon the systole of the auricle, and not upon that of the ventricle, which they distinctly preceded.

The next point of interest in this case was that the presystolic murmur persisted *per se* so long as the myocardium retained its normal tonicity; so soon, however, as the pulmonary congestion became sufficient to determine great secondary dilatation of the right ventricle with general venous remora, and consequent interference with the metabolism of the myocardium, then the presystolic murmur dwindled to a mere presystolic thump preceding a systolic mitral murmur, which at this stage was heard to pass into an impure first sound about the middle of the infra-axillary space as the stethoscope was moved to the left in the plane of the apex beat. When the weakness of the cardiac muscle became extreme, as happened during the last few weeks of this patient's life, even this thump disappeared, and a pure systolic murmur alone remained. This systolic murmur differed somewhat in pitch in the tricuspid and in the mitral areas, but it could be carried from the mitral area right round to the lower angle of the scapula without any break or alteration of its pitch. This systolic murmur was not solely due to tricuspid regurgitation,<sup>1</sup> because appropriate treatment was at first able to restore the tricuspid first sound more or less perfectly, but it did not restore the mitral first sound; the most it could do in this direction was merely to bring back the presystolic thump preceding the systolic murmur in the mitral area, and an impure first sound in the middle of the infra-axillary space. We are often more successful in this matter. A patient may come under treatment with a systolic murmur over the left apex, or it may

<sup>1</sup> In the case of John M'Owen, first admitted to Ward IV. on 10th December 1878, and subsequently repeatedly under observation, I satisfied myself that this is true, and that though it is a plausible idea to refer the systolic murmur in such cases to the right side alone, and its progress to the left to the gradual dilatation of the right ventricle, this is not really the case. M'Owen had a remarkable pigeon-breast, and his left apex never left the chest wall, yet after a time he had only a systolic murmur which could be carried right round to the scapula.



be over both right and left apices, and by appropriate treatment we first bring back a first sound to the right apex and with that a thump preceding the systolic murmur in the mitral area, and by and by, as the myocardium regains strength, there is a distinct presystolic murmur developed,<sup>1</sup> followed by a mitral systolic murmur, or by the usually accentuated first sound. In the case now under consideration there were only temporary and very imperfect attempts at closure of the right ventricle, and there never was any reproduction of the presystolic murmur after its disappearance. It seems of some importance to inquire why it is so much easier to produce comparative rehabilitation of the myocardium in some cases of mitral stenosis than in others. The answer to this seems to be primarily that the greater the degree of stenosis the more difficult it is to get any improvement in the metabolism of the myocardium, upon which the success of our treatment alone depends; and, second, that the earlier in life stenosis has occurred the more the development and nutrition of the heart is hampered. It is evident that in this matter the degree of stenosis is all-important.

It is obvious that when blood is detained in the lungs by a stenosed condition of the mitral opening, the intrapulmonary blood-pressure rises *pari passu* with the increasing congestion. So long as the myocardium retains its normal tone, the right ventricle is able more or less successfully to resist the dilating force acting upon its interior, even although that dilating force is sufficient, as in this case, to produce considerable dilatation of the pulmonary artery, which probably commenced long before any great or permanent dilatation of the tricuspid valve. The right auricle, it is true, is very early affected, and consequently there are few cases of mitral stenosis in which we cannot detect increased dulness to the right of the sternum, in the

<sup>1</sup> In Case I., p. 50, we have an instance in which a presystolic murmur became developed as the myocardium regained strength.

auricular region—the plane of the fourth rib. But so long as the tricuspid valve acts normally, this distention of the right auricle interferes but little with the carrying on of the circulation, and such cases often continue for years to present but few, if any, symptoms of the serious disease with which they are affected. But no sooner does the myocardium lose its tone than all this is changed, and serious cardiac symptoms at once set in. In this climate this loss of tone is most commonly brought about by an attack of bronchitis or feverish catarrh,<sup>1</sup> and in quite recent days that mysterious complaint influenza has had a most widespread and injurious effect in this direction, and we constantly find patients in whom the mitral stenosis was either congenital or dated from early infancy ascribing their illness to some catarrhal affection of quite recent date. The loss of vital tonicities of the myocardium involves an inability to cope with the normal and still less with any abnormal increase of the blood-pressure,<sup>2</sup> and this leads to dilatation of the cardiac cavities.<sup>3</sup> Hence a severe attack of bronchitis is not infrequently accompanied by tricuspid regurgitation. And bronchitis engrafted on a lung or lungs already congested through obstruction to the onward flow of the blood by a stenosed mitral opening exerts a most disastrous influence upon the muscle of the right ventricle, already slowly yielding before the abnormally high intrapulmonary blood-pressure. Dilatation of the right ventricle and regurgitation through the tricuspid opening occurring in these circumstances are fraught with ominous import to the safety of the patient. Hitherto he has been gliding along amid the calm if treacherous stillness of a mute disease, now he is plunged into the midst of those rapids where, with but few quiet intervals, he must for the future do battle for his life.

<sup>1</sup> *Vide* Lectures VI. and VII.

<sup>2</sup> *Vide* Lecture VI.

<sup>3</sup> *Vide* *The Senile Heart*, p. 220 and p. 40; also Roy and Adami, *op. cit.* p. 213.

Whenever the mitral opening is constricted the blood gradually though slowly accumulates behind the stenosed opening, and it is to this slow transference of the blood from the arteries to the veins, and the equally gradual impairment of the metabolism and consequently of the energy of the myocardium, that even in the most favourable cases death is ultimately due from asthenia, accompanied and indicated by the gradual accumulation of serum in the cavities of the body. It can only be in the rarest of cases, if ever, that this natural result of mitral stenosis is not precipitated by the intercurrent of some febrile ailment in the manner just referred to. A dilated right ventricle, the result of pyrexial relaxation coupled with the remediable congestion of a simple bronchitic attack, the heart being otherwise normal, is readily recovered from. But a persistent cause of increased intra-pulmonary blood-pressure, such as that due to limitation of the area of the pulmonary capillaries by destruction of the air-cells in emphysema, induces a permanent and incurable dilatation of the right ventricle. And we can readily understand that any permanent cause of pulmonary congestion, such as we have in mitral stenosis, must have a similar effect in preventing rehabilitation of the dilated right ventricle in exact proportion to the degree of stenosis present. In many cases of extreme mitral stenosis we have also another alteration of the vascular system (hypoplasia of the aortic system), which still further blocks the onward flow of the blood and hastens the rapidity with which the blood accumulates in the veins, increasing the readiness with which compensation may be ruptured, and which is itself a certain indication that stenosis has occurred at an early period of life. This makes us regard mitral stenosis commencing in early life as much more serious than when it happens later; and the earlier in life we discover mitral stenosis so much the graver must our prognosis be, even when it is not congenital but can be definitely connected with a preceding rheumatic attack.

As we shall presently see, the first effect of a rheumatic attack is pyrexial relaxation of the myocardium, and as the result of this we have a murmur of regurgitation frequently audible in the mitral area, more often only to be heard in the auricular area,<sup>1</sup> but of a curable character, and which disappears naturally or under treatment at the close of the attack. But we may also have an endocarditis of an acute, sub-acute, or chronic character, of rheumatic origin, which shrivels, thickens, and often mats together the fibrous tissues of the mitral cusps and of the *chordæ tendineæ*. This endocarditis occasionally starts in the early stages of the pyrexia, or it may follow it more or less slowly, with or without an intervening period of apparent cardiac health. The inevitable termination of such an endocarditic attack is more or less stenosis of the mitral opening. As the cardiac tissues are thus affected by rheumatism in a twofold manner, so we may have a curable form of mitral regurgitation which passes off as convalescence is established, or it may persist with more or less of cardiac pain and uneasiness till it terminates in stenosis of the mitral valve. At other times the curable regurgitation passes off, and there may be no symptom present beyond a little cardiac irritability; yet after the lapse of a year or eighteen months the signs of mitral stenosis will be found developing. When this result of rheumatism occurs in adult life, this stenosis follows the usual course and has attached to it the ordinary prognosis of mitral stenosis. But should a rheumatic attack in childhood or early youth be followed by any considerable constriction of the mitral opening, the effect of this is to dam the blood behind the constricted opening, to diminish the size of the blood-wave sent onwards, and thus to lessen the normal distention of the aorta, and to impair the metabolism and stunt the growth of all the tissues. In this way we have produced that arrested development so well known as the result of serious cardiac disease of an obstructive character occurring in early life, technically

<sup>1</sup> *Vide* Lecture VI.



termed hypoplasia, affecting primarily the aortic arterial system, and secondarily all the organs of the body. Of this we had a moderate example in this patient (Ormiston), in whom the aorta measured only half an inch in diameter. But this hypoplasia is occasionally much more marked; Allan Burns relates one instance in which the aorta barely admitted the little finger.<sup>1</sup> So extreme a degree of hypoplasia of the aortic system points to the development of stenosis of the mitral opening at a very early period of life; it may be even to a congenital stenosis of this opening, as Allan Burns has supposed.<sup>2</sup> The occurrence in some cases of an extreme degree of mitral stenosis with marked hypoplasia of the aortic system, with stunting of the growth of the body generally as well as of every organ in it, quite apart from any trace of a history of rheumatism or of any known cause of endocarditis, certainly favours this supposition.<sup>3</sup> Be this as it may, the important point for us to consider is that this hypoplastic condition of the aorta increases very materially the difficulty of rehabilitating the right ventricle, once this becomes dilated, presenting as it does a second and most material obstacle to the onward progress of the blood, which strengthens and

<sup>1</sup> This patient was a girl, delicate from birth, of nineteen years of age; the mitral valve was a tendinous septum, rigid and in some parts ossified; at its centre there was a puckered opening barely large enough to admit the tip of the little finger. Vide *Observations on some of the most Frequent and Important Diseases of the Heart*, by Allan Burns (Edinburgh, 1809), p. 32.

<sup>2</sup> His sixth species of congenital malformation of the heart consisted of those cases where the mitral valve is malformed, leaving merely a small opening leading from the left auricle into the ventricle; *op. cit.* p. 12.

<sup>3</sup> Virchow, in his pamphlet *Ueber die Chlorose und die damit zusammenhängenden Anomalien im Gefäßapparate* (Berlin, 1872), pp. 18, 19, attributes the valvulitis affecting chiefly the mitral, but occasionally also the aortic valves, in cases of aortic hypoplasia to the irritating results of intravascular pressure, the ventricle having more blood to send on than the aorta can receive. The usual result of such a state of matters is to produce cardiac dilatation in older people; there seems no reason why it should act differently in younger patients. Besides there seems a little difficulty in accounting for a superabundance of blood in a hypoplastic organism. On the other hand intra-uterine or infantile endocarditis ending in stenosis of the mitral opening must inevitably lead to hypoplasia of the aorta and of all the organs to which it is distributed, by cutting off the nutritive pabulum during the period of development.

reinforces that already existing in the stenosed condition of the mitral opening. Hence we see that the earlier the period of life to which the origin of mitral stenosis can be traced, so much the more readily may dilatation of the right ventricle and tricuspid regurgitation be produced by even comparatively slight secondary causes, and so much the more serious must be our prognosis, because under such circumstances it is never possible perfectly to rehabilitate the right ventricle, and the necessarily fatal transference of the blood-pressure from the arteries to the veins is inevitably hastened.

It will be remembered, then, that mitral stenosis is a common result of rheumatism, and that some degree of it is invariably present whenever the mitral valve has become diseased as the result of endocarditis of a rheumatic or a non-rheumatic origin. Whenever this stenosis occurs in infancy or early childhood, some degree of aortic hypoplasia is probably always concomitant, as it invariably is in those not infrequent cases in which mitral stenosis is congenital, whether as the result of intra-uterine endocarditis or simply of imperfect development.

Further, it will also be remembered that this stenosed condition of the mitral opening is to be recognised by the occurrence of a rough murmur preceding and running up to the apex beat and the carotid pulse, which is pathognomonic of this lesion. This murmur may, and often does, commence as a somewhat musical diastolic murmur following the second sound and growing rough towards its termination. This murmur is sometimes to be heard over the whole cardiac area, more usually, especially as to the strictly diastolic portion, just over the mitral valve where the fourth rib joins the sternum. Any portion of this prolonged murmur may be absent, and it may either be continuous or broken by a pause having the rhythm, though not the duration, of the normal soundless interval (second silence) of the cardiac cycle. The length of the pause varies with the amount of stenosis present, and may be accepted as an exact indication of this—the shorter the pause, the greater the stenosis. A purely mitral

diastolic murmur is of course unaccompanied by any signs of aortic regurgitation ; it has a clear aortic second preceding it, and is accompanied by no jerking of the pulse produced or increased by elevation of the arm, and by no dilatation of the left ventricle evinced by depression of the apex beat. This murmur is more or less prolonged and frequently slightly musical in character, and is chiefly to be heard in the mitral area or just over the mitral valve. A short, soft, simply blowing murmur, even though only audible at the sternal end of the fourth rib, must always be viewed with suspicion as probably always of aortic origin. Cases of pulmonary regurgitation are so rare that they can scarcely be regarded as in any way likely to complicate the diagnosis or to throw any difficulty in its way. Such cases are chiefly of congenital origin ; there are only a few cases on record, and in none have any signs been observed in any way likely to obscure the diagnosis of a mitral diastolic murmur.<sup>1</sup> Extreme irregularity of the pulse either with or without pyrexia is very suggestive of the presence of mitral stenosis, and the probability of this is increased if there is an audible thump preceding or accompanying the apex beat ; if there is hæmoptysis, and if there are signs of enlargement of the right side of the heart, and reduplication or accentuation of the second sound, most distinctly audible at the third left costal cartilage, the accentuated second being always the pulmonary.

A simple systolic apex murmur of mitral regurgitation is occasionally the sole indication of mitral stenosis, but in such cases this murmur is preceded by a more or less evident thump, and it generally ceases at or about the middle of the infra-axillary space, being there replaced by a more or less impure first sound. In those cases in which the systolic murmur can be carried right round to the angle of the scapula

<sup>1</sup> At pp. 999 and 1000 of his *Diseases of the Heart and Aorta* (Dublin, 1875), Dr. Hayden has published notes of four cases of regurgitation through the pulmonary valves, which confirm the view taken in the text that even apart from the rarity of such cases there is but little risk of confusion likely to arise from the signs themselves.

we invariably have a heaving impulse at the lower part of the sternum, and over this another systolic murmur of a different pitch, pointing to considerable dilatation of the right ventricle with tricuspid incompetence, confirmed by the



FIG. 15.

presence of marked hepatic pulsation. In all cases of mitral stenosis there is very generally a distinct pulsation visible in the auricular area above the fourth rib<sup>1</sup> and to the left

<sup>1</sup> Fig. 15 gives an excellent representation of a large auricle lying along the anterior base of the heart, as it not infrequently does in mitral stenosis. Vide *Journal of Pathology and Bacteriology* (July 1896), p. 75.



of the sternum; this pulsation is either wanting or it is at least inconsiderable in cases of simple regurgitation in which the valve is unaffected. A systolic murmur is often to be heard in this auricular area as an indication of regurgitation in mitral stenosis; and indeed a murmur in this area is generally one of the earliest indications of regurgitation from any cause whatever.

The prognosis of mitral stenosis is favourable so long as the right side is unaffected, and the rough presystolic murmur persists; this favourable prognosis being subject to modification according to the circumstances of the patient, but especially according to the period of life at which the stenosis originated, being more favourable the later in life this has happened. But whenever the right ventricle becomes secondarily dilated the prognosis at once becomes serious, and all the more serious the earlier in life this occurs, because the probability is then greater that the stenosis dates from early childhood or infancy and the co-existence of hypoplasia of the aortic system is more certain. But the coincidence of dilatation of the right ventricle with hypoplasia of the aortic system at once makes the prognosis most grave, and reduces the expectation of life from many to only a few years, except under the most favourable circumstances.

Sufferers from mitral disease are not liable to sudden death from heart failure, like those labouring under aortic incompetence; they die normally from asthenia preceded by dropsy. Many pass through a long life with comparatively little inconvenience; most, however, suffer a good deal from breathlessness, palpitation, and irregularity of the heart's action, the latter being often extremely persistent and annoying during the latter years of life. Cerebral embolism is an occasional cause of sudden death in cases of mitral stenosis, but pulmonary embolism is a much more frequent and quite as serious an accident, though death in these circumstances is never so sudden. Ruptured compensation from various causes is much more common in mitral than in

aortic disease, but it is of much less serious import and is more easily recovered from, except under the special circumstances already referred to.

The treatment of such cases must be purely tonic. While compensation is perfect, nutritious diet and the avoidance of all over-exertion are all that is required, though arsenic and iron, or arsenic and strychnine may be usefully combined. When compensation is ruptured, tonic doses of digitalis are called for, and if extreme irregularity is present large doses of digitalis for a time are of great service, though even this form of heart trouble yields to moderate doses of digitalis if they be conjoined with a light but nutritious diet and abundance of rest. The great object of treatment is to maintain and, when needful, restore the tone of the myocardium. Even when there is considerable hæmorrhage from pulmonary infarction it is seldom that any other treatment is required, though an embolic pneumonia is much benefited by the conjunction of moderate doses of chloral with the digitalis.

As we never have mitral stenosis without some degree of regurgitation, so some hypertrophy of the left ventricle is generally present, but it is of slow growth and is never excessive. Marked dilatation of the left ventricle is less common and depends upon pyrexial relaxation of the myocardium, or increased obstruction to the circulation from arterial atherosclerosis, or secondary cirrhosis of the liver or other organs, the result of venous remora.

## LECTURE VI

### ON CURABLE MITRAL REGURGITATION ; ITS VARIOUS CAUSES, THE PHYSICAL SIGNS BY WHICH IT IS REVEALED, AND ITS TREATMENT

IN a former lecture<sup>1</sup> I pointed out that the only unequivocal proof of the actual existence of disease of the mitral valve is the presence of an auricular-systolic, or, as it is commonly termed, a presystolic murmur, or at all events proof that such a murmur has been heard at some former period of the patient's life ; for, though this murmur frequently disappears or alters its character, the lesion upon which it depends is permanent. It would have been more correct to have said that the only unequivocal proof of disease of the mitral valve is the determination of a constriction of the auriculo-ventricular opening, however that may be established, for stenosis of that opening is the usual condition when the mitral valve is diseased, and though it is always present when a presystolic murmur is to be heard, it also frequently exists when that murmur is not only absent at the time of examination, but has never been recognised at any past period of the history of the case, though even in these instances the presence of stenosis is frequently revealed by certain signs which have been already described.<sup>2</sup> As in the lecture referred to, however, I alluded chiefly to the character of the murmur distinctive of disease of the mitral valve, the statement may be accepted as correct to that extent. At present

<sup>1</sup> Lecture IV. p. 110.

<sup>2</sup> Lecture V. p. 135.

I intend to refer to mitral regurgitation unaccompanied by disease of the mitral valve, and shall have to traverse cursorily a wide series of diseases in which this lesion is found more or less frequently. In a large proportion of these cases it is happily permanently curable, though revealed by what to many even yet seems the certain sign of incurable organic mischief—a systolic mitral murmur. The importance of this observation needs no comment. To the patient it involves the issues of life and death, a comparatively short illness, and thereafter a normal and, it may be, a long and healthy life; or permanent ill health, an abnormal life, and shortened days. To the physician it brings home the paramount importance of an accurate diagnosis, and substantiates in a most striking manner the value of appropriate treatment.

The vigour of any muscle depends upon the perfection of its metabolism, and as the heart is not only able to originate spontaneous rhythmic movements but also to store up a large reserve of energy, its metabolism is of a much higher character than that of the ordinary skeletal muscles<sup>1</sup> and is readily affected by any changes in the quantity or quality of the blood which furnishes its basis. Hence pyrexial diseases or such as interfere with the general metabolism exert an injurious effect upon the myocardium, and impair the discharge of its function.<sup>2</sup> But the function of the heart is to maintain the circulation by contracting on the blood within its cavities and forcing it onwards, and whatever impairs this function leads to residual accumulation within the ventricles and to gradual dilatation of their cavities.<sup>3</sup> The result of even a slight dilatation is the establishment of so-called

<sup>1</sup> Foster's *Physiology* (1883), p. 344.

<sup>2</sup> Old hearts as well as young ones are liable to have their function impaired by similar causes, but are not often curable. For an account of the results of impairment of function on them, vide *The Senile Heart*, A. and C. Black: London, 1894.

<sup>3</sup> "The amount of residual blood (in the ventricle) is always increased whenever, the aortic pressure remaining constant, the force of the ventricular contraction is reduced from malnutrition or other causes."—*Vide* Roy and Adams, *Philosophical Transactions*, vol. 183, p. 213.



relative insufficiency, not because the auriculo-ventricular opening itself is dilated—that does happen, but at a later period of the affection—nor because the segments of the valve are unable to close the opening, as one alone of these segments is almost sufficient for this purpose; but because, owing to the separation of the cardiac walls, by dilatation of the cavity, the insertions of the *chordæ tendineæ* into the papillary muscle are set so wide apart and so far from the centre of the ventricle that the trifling pressure of the auricular blood is unable to bring the valve-segments into apposition just before the commencement of the ventricular systole.<sup>1</sup> But any interference with the instantaneous closure of the valve at the moment of ventricular systole favours regurgitation,<sup>2</sup> and in these cases this is a constant phenomenon, varying in degree at different times.

In all these cases there is a more or less impure first sound audible in the mitral area, or a transient systolic whiff may terminate in an apparently normal first sound, and in still other cases a murmur is heard throughout the whole of the systole from its commencement to its end.

This is quite in accordance with the account just given. In slight dilatation at the commencement of the ventricular systole the valve-segments are not in apposition as they ought to be, and there is some regurgitation, but this ceases as the systole progresses and the valve-segments are perfectly closed. When the dilatation is greater the valve-segments never come together, and the regurgitation persists throughout the whole of the systole.

At a very early stage of its dilatation the right ventricle forces the apex from the chest wall into the thoracic cavity. How slight is the dilatation necessary for this may be learned by placing a finger on the apex and stopping the respiration. Even during the few seconds we can hold our breath the

<sup>1</sup> *Vide* Ludolph Krehl, *Archiv für Anatomie und Physiologie* (Leipzig, 1889), S. 291.

<sup>2</sup> *Vide* Pettigrew, *Transactions of the Royal Society of Edinburgh*, vol. xxiii. p. 796, note.

apex will be found to have receded so far as to be almost imperceptible. When this dilatation of the right ventricle is from a more permanent cause, it is readily recognised by the more forcible beat communicated through the stethoscope applied over the lower part of the sternum long before the development of any tricuspid murmur. This altered relation of the apex to the chest wall, coupled with the probable low tension and comparatively trifling force of the fluid veins formed at the mitral orifice at the commencement of ventricular systole, are sufficient to account for this murmur never reaching the mitral area otherwise than as at the most a slight impurity of the first sound at this stage. On the other hand, there are but few exceptions to the rule that no sooner have we any certain indications of even slight dilatation of the left ventricle than we are able to pick up a faint but distinct systolic murmur in the auricular area, due to the impingement of these fluid veins upon the auricular wall, and the communication of their vibrations to the anterior chest wall, with which the appendix of the left auricle is in immediate contact. As I have already stated, it is chiefly in connection with mitral stenosis that the appendix of the left auricle attains so great a size as to lie round the base of the heart in close juxtaposition to the anterior chest wall,<sup>1</sup> but we know from Walshe that an inspection carried on for many years of the bodies of male subjects cut off by various diseases had convinced him that in almost every instance the appendix of the left auricle is visible anteriorly, as the organs lie *in situ*.<sup>2</sup> Walshe says that the middle point of the auricle in this position from above downwards corresponds to the cartilage of the third rib (*vide* frontispiece). This is the position in which a systolic murmur is generally to be heard, and a systolic pulsation occasionally to be felt, as an early indication of commencing dilatation. We know that the left

<sup>1</sup> As figured at p. 164; *vide* also *Journal of Pathology and Bacteriology* (Edinburgh and London, July 1896), p. 75.

<sup>2</sup> *Diseases of the Heart and Great Vessels*, 3rd edition (London, 1862), p. 2.

edge of the sternum divides the pulmonary artery; one-half lies beneath the sternum, and the other in the second interspace. If we place the tip of the finger in the second interspace close to the sternum so as to cover the pulmonary artery, the position of maximum intensity of this auricular murmur will be found to lie just to the left of the finger. There are comparatively few cases in which the *appendix auriculi* is too short to reach the chest wall, even when slightly dilated, and there are still fewer in which an elongated appendix gets twisted backwards, and so prevented from reaching the chest wall. In all these cases auricular pulsation and an auricular murmur are naturally wanting. Pulsation in the auricular area is sometimes great and well-marked, so much so that a late distinguished professor and teacher of clinical medicine upon one occasion sent me a case of chlorosis with auricular pulsation as one of aortic aneurysm.<sup>1</sup>

Skoda was the first to point out that when the right ventricle was dilated and hypertrophied as the result of mitral disease a systolic murmur was often to be heard, as he supposed, in the pulmonary artery. This he ascribed to softening of the lining membrane of the artery.<sup>2</sup> Joseph Meyer has shown that this hypothetical alteration of the lining membrane of the pulmonary artery in such cases is a pure myth, and he has attempted to explain this peculiar phenomenon by another equally untenable supposition.<sup>3</sup> The murmur itself is well known, and has been described and variously explained by many writers on the subject,<sup>4</sup> but it was reserved for Naunyn to point out that a murmur of this character, whether it accompanies a systolic murmur in the mitral area, or is itself the sole indication of mitral regurgitation, has its position of maximum intensity not in the

<sup>1</sup> Vide *Lancet* (September 1877), p. 386.

<sup>2</sup> *Abhandlung über Perkussion und Auskultation*, 3. Auflage (Wien, 1844), S. 223.

<sup>3</sup> Virchow's *Archiv*, Bd. iii. Heft 2, S. 277.

<sup>4</sup> By Friedreich, *Krankheiten des Herzens*, 2. Auflage (Erlangen, 1867), S. 173; Bamberger, *Lehrbuch der Krankheiten des Herzens* (Wien, 1857), S. 92; Von Dusch, *Lehrbuch der Herzkrankheiten* (Leipzig, 1868), S. 205, etc.

pulmonary area, but to the left of it, where the appendix of the left auricle comes up from behind. Naunyn's explanation is the only one consistent with the facts, and is now universally received.<sup>1</sup>

Daily experience in the sick-room has long since taught us that all pyrexial diseases are accompanied by muscular relaxation and debility, and from the experiments of Roy and Adami we know that any increase in the aortic blood-pressure, or, the aortic pressure remaining constant, any diminution in the force of the ventricular contractions results in increase of the residual blood and ultimate dilatation of the ventricle.<sup>2</sup> When the gradual wasting of the tissues—as in phthisis or carcinoma—is accompanied by a diminution of the mass of blood, the heart simply atrophies in all its dimensions, and its cavities are correspondingly diminished in size (concentric atrophy).<sup>3</sup> But in other complaints, whether associated with pyrexia or debility, the heart is liable to undergo slow and gradual dilatation by the agencies just referred to, provided the morbid influences are long enough maintained. I have elsewhere described the many interesting and peculiar symptoms associated with failure of the senile heart,<sup>4</sup> and I shall not further allude to them here, except to remark that some of these are now and then engrafted on younger hearts, partly because age is a matter of tissue change and not of years, and partly because some of those symptoms are liable to affect weak hearts at any age, though they are more commonly observed after middle

<sup>1</sup> "Ueber den Grund weshalb hin und wieder das systolische Geräusch bei der mitral Insufficienz am lautesten in der Gegend der Pulmonalklappe zu vernehmen ist," *Berliner klinischer Wochenschrift* (1867), No. 17, S. 189; vide also Paul Niemeyer, *Handbuch der Percussion und Auscultation* (Erlangen, 1870), Bd. ii. Abtheil i. S. 140, and Gerhard's *Lehrbuch der Auscultation und Percussion*, 2. Auflage (Tübingen, 1871), S. 283.

<sup>2</sup> *British Medical Journal* (December 1888), p. 1320, etc; also *Philosophical Transactions*, vol. 183, p. 213.

<sup>3</sup> Walshe considers that the sustainment of the heart at par in those cut off by such wasting diseases must be regarded as the equivalent of hypertrophy, *op. cit.* p. 274.

<sup>4</sup> *The Senile Heart, its Symptoms, Sequelæ, and Treatment.*



life. Neither shall I make here any special reference to cardiac dilatation due to pyrexial disease, as it arises in a similar manner, is revealed by similar signs, and follows a similar course to that occurring in connection with more chronic diseases in which the various stages are more readily investigated.<sup>1</sup> In former days cardiac murmurs were divided into organic and functional; the former were recognised as dependent on irremediable valvular lesions, the latter were supposed to occur independently of any alteration of the organ. Functional, or inorganic, murmurs were classified under two heads—hæmic and dynamic. A hæmic murmur is one supposed to be produced by the passage of abnormal blood through a perfectly normal heart. It is always systolic in rhythm, and has been regarded by most observers as always basic in position. A dynamic murmur, on the other hand, though always systolic in rhythm, is apical in position and regurgitant in character, and is supposed to be produced by a perturbed or irregular action of the cardiac muscle, quite independent of any abnormality of the blood, which, however, always co-exists.

Chlorosis and the chlorotic cardiac murmur is the typical hæmic murmur; chorea and the apex murmur that so often accompanies it is the typical dynamic murmur. In chlorosis the blood lesion is primary and paramount; in chorea the blood lesion is essentially secondary to pyrexia, and specially to the exhaustion that follows the continuous morbid jactitation so characteristic of this disease. Now we know that the so-called functional murmurs of both kinds essentially depend upon dilatation of the heart due to residual accumulation from imperfect discharge of its function by a heart unable, through debility, to cope with the blood-pressure which is either natural or only slightly raised. As the development

<sup>1</sup> In severe and fatal typhus the heart was sometimes found to be as "limp as a piece of wet rag." Quoted from Louis by Stokes, *Diseases of the Heart and Aorta* (Dublin, 1854), p. 368. In less serious cases a systolic murmur was frequently developed, as in other pyrexial diseases, during the course of the fever, *op. cit.* p. 423.

of this affection is essentially similar in all the various diseases with which it is found connected, and as in chlorosis this development is more gradual and can be more readily followed, it will be most instructive to consider the rise and progress of the chlorotic cardiac murmur.

It is unnecessary to enter particularly into the pathology of chlorosis; it is sufficient to describe it as a form of spanæmia of common occurrence, especially among country girls who, for the first time, find themselves exposed to the unhealthy surroundings inseparable from domestic service in a town, and in whom the prominent derangement of the genital function is amenorrhœa. It is also common enough in town-bred girls of a better class, especially about the age of puberty, and in them the menstrual discharge is pale and usually scanty, menorrhagia being exceptional, and always followed by great exhaustion. The lips of such patients may be rosy, and the cheeks present a certain amount of youthful bloom, but the interior of the lips, the gums, and especially the conjunctivæ of the lower eyelids, are pale and bloodless. The face inclines to be puffy rather than clear cut or sharp in its outlines; the ankles are slightly œdematous; the appetite is irregular and capricious, and there is breathlessness and palpitation on the slightest exertion. This condition is also signalised by the constant occurrence in the veins of the well-known *bruit de diable* or humming-top murmur; this venous murmur is continuous and audible during both systole and diastole, its intensity increasing with thoracic and cardiac aspiration, and diminishing with expiration and the cardiac systole, so that the venous hum waxes and wanes as these alternate.

This venous murmur is caused by the production of Savart's fluid veins<sup>1</sup> at the point where it is heard, and it is readily produced in spanæmic individuals by moderate pressure over any vein of tolerable size, such as the external jugular. It is also readily heard over the eyeball, over the

<sup>1</sup> Lecture I. p. 46.

*torcular Herophili*, over the upper part of the innominate veins, especially the right one, and over other parts of the venous system where there exists naturally, what is produced artificially by pressure, a flow of blood through a part relatively constricted to that beyond it. As this venous murmur is readily heard, in appropriate circumstances, in certain positions, such as the *torcular Herophili*, where there is a normal relative constriction insufficient to produce sonorous veins, there can be little doubt that the increased narrowing of the opening is due to increased friction between the wall of the vein and the layer of spanæmic blood next it, leaving uninfluenced the axial portion of the stream in which these fluid veins are formed. Besides an appropriate condition of the vessel, and of the blood, a certain amount of current force is needful to produce these sonorous veins; hence they are often inaudible in the recumbent posture, and become at once audible when the force of the blood current is increased by elevating that part of the body at which we are listening. Many observers ("London Heart Committee," Liman, Winterich, etc.) have pointed out that a venous murmur is often to be heard in those who appear to be in perfect health, but in those individuals this murmur is altogether wanting in that loud and sonorous character so remarkable in well-marked cases, and all that this observation proves is that a certain amount of spanæmia is not inconsistent with apparent health.

But the venous murmurs that accompany chlorosis are of much less interest and importance than those heard within the cardiac area. These cardiac murmurs are always systolic in character, but their position of maximum intensity has been located on various parts of the cardiac area by different observers. The classic position of the chlorotic murmur is the aortic orifice, as narrated by Hope,<sup>1</sup> Bellingham,<sup>2</sup> Potain,<sup>3</sup>

<sup>1</sup> *A Treatise on Diseases of the Heart* (London, 1839), p. 106.

<sup>2</sup> *Diseases of the Heart* (Dublin, 1853), p. 137 and p. 142.

<sup>3</sup> *Dictionnaire Encyclopédique des Sciences Médicales*, tome iv. p. 392.

Marey,<sup>1</sup> and others, but the aortic origin of the chlorotic murmur in its early stage is absolutely negated by the fact that at this stage it is not propagated along the course of the arteries; as Hayden has said, "it has no definite line of propagation."<sup>2</sup> Parrot<sup>3</sup> has supposed that this murmur is always of tricuspid origin, and Stark<sup>4</sup> has shown that it may be of mitral as well as of tricuspid origin. Most observers are, however, agreed that the cardiac murmur heard in the early stage of chlorosis is basic in position and has "no definite line of propagation," while its position of maximum intensity is the neighbourhood of the pulmonary artery, where it is always best marked and often only to be heard. So usual indeed and so well defined is this position of maximum intensity that Marshall Hughes<sup>5</sup> has suggested the pulmonary artery itself as the source of this murmur. But no murmur of strictly pulmonary origin could possibly be referred to all the four cardiac orifices in turn, as has been the case with the chlorotic murmur; and indeed careful observation teaches us that this murmur, though audible in the pulmonary area, has its position of maximum intensity not there, but in the second interspace just to the left of the pulmonary artery, exactly where the appendix of the left auricle comes up from behind. It is in fact an auricular murmur, a murmur of mitral regurgitation audible in the auricular area (*vide* p. 171), and its further development is strictly in accordance with the ordinary mode of evolution of such murmurs in the course of progressive dilatation of the heart.<sup>6</sup> In chlorosis we have first the *bruit de diable*—the

<sup>1</sup> *Physiologie Médicale de la Circulation du Sang* (Paris, 1863), p. 479. Marey reverted to this view apparently because it suited with his idea that a low blood tension in the aorta favours the formation of fluid veins at its orifice. It is rather unfortunate for this theory that this murmur is often only to be heard in the neighbourhood of the pulmonary artery, where an accentuated second sound assures us that the blood tension is always increased.

<sup>2</sup> *Op. cit.* p. 252.

<sup>3</sup> *Archives Gén. de Médecine*, 6ième série, tome viii. 1866; vol. ii. p. 158.

<sup>4</sup> *Archiv der Heilkunde* (1863), p. 47; and *Gaz. Hebdom.* (1863), p. 262.

<sup>5</sup> *Guy's Hospital Reports*, second series, vol. vii. p. 161.

<sup>6</sup> *Vide The Senile Heart*, p. 56.



venous hum of serous polyæmia—chiefly audible in the right jugular vein; next we get accentuation of the pulmonary second, as the earliest indication of obstruction to the circulation from residual accumulation in the ventricles; and accompanying this accentuated pulmonary second, or speedily following its development, a systolic murmur in the auricular area, where a distinct pulsation is very often both to be seen and felt (*vide* p. 164). This auricular murmur radiates all round its position of maximum intensity, but, owing to the resonant properties of the sternum, it is most distinctly propagated to the right in the direction of the aorta, but it is never propagated into the arteries. Accompanying this auricular murmur there is always an impure first sound in the mitral area, and this frequently passes into a systolic murmur. There is always more or less pulsation at the lower end of the sternum, with more or less evident undulation or pulsation of the jugular veins, indicating engorgement and dilatation of the right ventricle, but a distinct tricuspid murmur is not a very usual phenomenon in spanæmic cases, unless we look upon the murmur audible in the mitral area in advanced cases as of tricuspid origin, which there are some good reasons for doing. In still more advanced cases, when the ventricles have become not only dilated but somewhat hypertrophied, we get a systolic murmur originating at the aortic orifice and propagated along the arteries, and no doubt the same thing happens at the pulmonary orifice, the murmur in each case being due to the large blood-wave sent on by the dilated ventricles coupled with the relative constriction at the arterial orifices due to the abnormal friction of the spanæmic blood.<sup>1</sup> We can seldom trace the development of these murmurs, because when a patient comes under treatment she gets cured and the evolution of the murmurs ceases. But in every case we can trace their gradual involu-

<sup>1</sup> Beau, *Traité d'Auscultation* (Paris, 1856), pp. 366 and 565; *vide* also "The Position and Mechanism of the Hæmic Murmur," *Lancet* (September 1877), p. 383, etc.

tion in the reverse order to that in which they have just been described as arising, the aortic murmur being the first to disappear, and every trace of the auricular pulsation and of the auricular murmur vanishes long before the venous hum ceases to be heard. Further, though the primary murmur is in most cases an auricular one, yet we know that there are exceptional cases in which the *appendix auriculi* does not reach the surface; in these cases there can be no auricular murmur,<sup>1</sup> and the murmur may be ascribed to any one of the four cardiac orifices at each of which in turn it presents itself with well-defined intensity. Thus the discrepancies in the views regarding the point of origin of this spanæmic murmur have obviously been based upon accurate observations imperfectly understood.

The cause of the cardiac dilatation is readily understood. The spanæmia of chlorosis is what Beau has termed a serous polyæmia, a state analogous to that produced by repeated venesections, in which the red corpuscles are diminished and the serum relatively, and in some cases actually increased. In this condition, as we can readily understand, there is great impairment of the cardiac metabolism and consequent reduction in the force of the ventricular contractions, with residual accumulation within their cavities, resulting in incompetence first of the mitral, and ultimately of both mitral and tricuspid valves. Beau found that repeated venesection produced in dogs and rabbits a spanæmic condition, to which he gave the name of serous polyæmia. He also found that, when bled to death, the hearts of those animals in whom serous polyæmia had been fully developed were not only larger, more dilated, but also weighed from one-fifth to one-sixth more than those of healthy animals killed by one fatal hæmorrhage from a severed carotid.<sup>2</sup> The dilatation is

<sup>1</sup> *The Senile Heart*, p. 55.

<sup>2</sup> *Archives Générales de Médecine*, 4ième série, tome ix. (Paris, 1845), p. 156. The following case shows that similar results follow a similar cause in man :— W. R. aet. 55, admitted to Ward XXXII., New Royal Infirmary, 27th April 1880, labouring under serous polyæmia of a serious character (pernicious

primarily compensated by the reserve force of the heart, and the hypertrophy is slowly developed by means of the agencies already referred to when speaking of a similar condition in connection with aortic incompetence (*vide* p. 84).

Beau,<sup>1</sup> Bamberger,<sup>2</sup> Friedreich,<sup>3</sup> Wunderlich,<sup>4</sup> and Stark,<sup>5</sup> have all noted that the heart in chlorosis is large—dilated and slightly hypertrophied—the serous polyæmia of disease having evidently a similar effect on the heart to that produced by hæmorrhage. The true aortic murmur of chlorosis is a late development, and the conditions on which it depends are more serious than those subsisting at an earlier period of the disease; still they are known and acknowledged to be curable. But dilatation and hypertrophy are not always curable in one set of conditions, and always incurable in another; the degree of curability depends upon the size of the heart, partly on the amount of dilatation present, more perhaps upon the degree of hypertrophy, to some extent upon the cause of the affection, and even more upon the patience and skill of the physician. If the cause be curable, the result may certainly be remedied by nature or more surely by art. I know many who years ago were believed by competent observers to labour under incurable disease of the heart, who for long have been perfectly free from any trace of such disease; in my own experience many have been rescued from such a condition by the skilful use of remedies, and I shall presently relate the histories of a few of these.

anæmia) the result of hæmorrhage from the bowels of long standing. All the murmurs of chlorosis were present in this case. He died 14th May 1880. On *post-mortem* examination the whole surface of the body was blanched and anæmic, rigor slight. The heart weighed 12½ oz.; its muscular fibre on both sides was flabby and anæmic, not fatty. The aortic valves were competent; the cone diameter of the aortic orifice was 1 inch, of the mitral 1·5, of the pulmonary artery 1·2, and of the tricuspid 2 inches. The left ventricle measured 3 inches in length; its wall from  $\frac{1}{4}$  to  $\frac{1}{2}$  inch in thickness. The right ventricle measured 4 inches in length; its wall  $\frac{1}{3}$  of an inch in thickness.

• All the organs were blanched; no special cause of death was found.

<sup>1</sup> *Op. cit.* p. 169; *vide* also his *Traité d'Auscultation* (Paris, 1856), p. 355.

<sup>2</sup> *Op. cit.* S. 83 and S. 246 (Stuttgart, 1856), Bd. iv. S. 354.

<sup>3</sup> *Op. cit.* S. 172.

<sup>4</sup> *Handbuch der Pathologie und Therapie.*

<sup>5</sup> *Archiv der Heilkunde* (1863), S. 47, and *Gaz. Hebdomadaire*, p. 262.

But to this hopeful view there is also a reverse, and it has fallen to my lot to see sufferers from incurable cardiac disease who, not many years previously, had been assured by most skilful physicians that they had only a functional ailment out of which they would grow. But so-called functional murmurs are always murmurs of dilatation, and, except under the most favourable circumstances, nature only too often fails to remedy this. Even skilful treatment may fail to completely restore the heart to its pristine condition, but it always arrests the progress of the complaint, and the benefits it confers are often more evident as years roll on. Many years ago I vainly endeavoured to procure the contraction of a flabby, dilated, chlorotic heart; in time and with my sanction she was happily married, and is now the mother of several healthy children. Her heart has given no trouble, and though a loud systolic murmur was long present, of late this has become less obvious and it is now only occasionally to be heard faintly.

The early recognition of those so-called functional murmurs, coupled with the more accurate knowledge we now have as to their causation, will doubtless in the future be the means of enabling us to avert the drifting into incurable disease of many of these cases, and to restore to usefulness many youthful lives that would otherwise have been doomed to sad despair.

In the person of Helen Christison, Ward XIII., Bed 9, admitted 12th February 1875, we have had an excellent opportunity of observing the gradual and consecutive disappearance of all the murmurs described, coincident with the restoration of the patient to perfect health.

In all chlorotic cases the central lesion, and, so far as the circulation is concerned, that upon which all the phenomena observed depend, is certainly an alteration of the blood revealed by a venous murmur.

Slight venous murmur is often to be found apart from any untoward sign, but when at all well marked it is



invariably associated with an auricular murmur, and consequently with some degree of cardiac dilatation. Murmurs in the mitral area of chlorotic origin are comparatively rare; in such cases there is usually little more than an impurity of the first sound in this area. Now and then, however, the murmur is exceptionally loud and distinct, and it may be impossible to decide—apart from the results of treatment—whether it is due to simple dilatation or to actual valvular lesion. The following case is a striking instance of this:—

CASE XVII. Elizabeth Heughson, a domestic servant, aged nineteen, admitted to Ward XIII. on 28th March 1873, first examined on 29th March. The patient is a middle-sized girl, of fair complexion and anæmic appearance. About two years and a half ago she suffered from acute rheumatism with pain over the cardiac area; since that time she has been liable to occasional rheumatic pains in various parts. She has been much exposed to cold and damp, from the condition of her dwelling at Oxford, and also from having to take part in the family washing. Her present illness came on in November last as simple weakness, which since then has been gradually increasing. She has not menstruated for the last two periods; menstruation previously had been scanty. Her digestion is feeble, and her abdomen flatulent; other systems normal except the circulatory. The heart's apex is felt to beat somewhat diffusely between the fifth and sixth ribs, two inches and three-quarters from the left edge of the sternum. In the parasternal line, one inch from the left edge of the sternum, cardiac dulness commences at the upper edge of the third rib and extends downwards to the liver dulness. Transverse dulness at the level of the fourth rib begins one quarter of an inch to the right of the sternum and extends across for a distance of five inches and one quarter. In the mitral area there is a loud, blowing, systolic murmur, which is propagated round the chest to beneath the angle of the scapula, and is followed by an accentuated second sound. In the aortic area the first sound is replaced by a murmur,

followed by a weakened second sound. In the pulmonary area the first sound is also replaced by a murmur, most distinctly heard one inch and a half from the left edge of the sternum in the second interspace; it is followed by a distinctly accentuated second sound. The pulse is 108, small and feeble. A loud venous hum is audible in the right jugular vein. She was treated with various preparations of iron, at first along with digitalis; latterly with the ammonio-citrate of iron alone. Under treatment she improved in strength, and regained a more healthy appearance; her palpitations almost entirely ceased, but her murmur remained unaltered. On the 23rd of May she was sent to the Convalescent Hospital, whence she returned for examination on the 19th of June. She was then found to have completely regained all the appearance of health, her cardiac murmurs had entirely disappeared, the accentuation of the pulmonary second was gone, and the venous hum had ceased. In spite of the distinct history of rheumatism, which made the prognosis doubtful, the cure was complete. The result of treatment showed that the valves had been quite unaffected, and that the whole of the very striking phenomena had been due to simple dilatation of the left ventricle, the result of chlorotic spanæmia. The complete disappearance of the accentuation of the pulmonary second sound is conclusive proof that here we had an actual cure by contraction of the ventricle, and not merely an example of the vanishing of a murmur, the lesion remaining.

I need not say that in cases of acute rheumatism, as in other pyrexial diseases, we frequently have the development of a systolic mitral, an auricular or so-called pulmonary murmur and a systolic aortic murmur—the mitral murmur sometimes alone, the auricular occasionally by itself, but the aortic murmur is never to be heard unless one or other, if not both, of the before-mentioned murmurs is also present. All these phenomena ordinarily disappear on the return of health. After what has been already said, this is readily understood. The

defective metabolism of the heart, resulting from the depraved, spanæmic condition of the blood, weakens its contractile force and causes residual accumulation in the ventricle. The resulting dilatation of the cavity separates the papillary muscles from its centre and prevents the *chordæ tendineæ* from bringing the valve segments into apposition at the moment of systole, hence regurgitation, varying in degree at different times, the murmur itself varying in position and intensity with the degree of regurgitation, as already pointed out.<sup>1</sup> When the normal healthy condition of the blood is restored cardiac metabolism again becomes perfect, the muscle recovers its healthy tone, the valve segments close normally, and all signs of a cardiac affection disappear. Should, however, the fibrous tissue of the valves or of the *chordæ tendineæ* become thickened or shrivelled as the result of the rheumatic process, then all the signs of incurable mitral disease become developed. Sometimes this follows consecutive to those of the curable form, the one set of signs passing uninterruptedly into the other. At other times the curable form of the disease is apparently perfectly recovered from, and the incurable form is subsequently developed after a varying interval of seeming health, sometimes as the result of the primary rheumatic affection, at others, more or less evidently caused by the persistence of rheumatic valvulitis in a chronic, sub-acute, or recurrent form.

Mitral murmurs of a curable character are frequently found in connection with chorea. The close connection that has long been recognised to subsist between chorea and rheumatism<sup>2</sup> enables us to understand that many murmurs originating during the course of chorea may become per-

<sup>1</sup> *Vide antea*, p. 169; also Roy and Adami, *Philosophical Transactions*, vol. 183, p. 213; and also Ludolph Krehl, *Archiv für Anatomie und Physiologie* (Leipzig, 1889), S. 291.

<sup>2</sup> In his *Contribution to Practical Medicine* (Edinburgh, 1862), p. 68, etc., Dr. James Begbie has well described the intimate connection between these two diseases, and has told us the history of the discovery of this connection. I myself have repeatedly seen patients in whom choreic movement alternated with rheumatic inflammation of the joints.

manent and incurable, but the larger proportion are curable and disappear with returning health. Such murmurs have been called dynamic, and have been ascribed to irregular action of some part of the cardiac muscle, the result of clonic spasm, and especially to clonic spasm of one or other of the *musculi papillares*, similar in character to those spasms which in this disease affect the voluntary muscles.<sup>1</sup> But clonic spasm of any part of the heart muscle, though it might account for an intermittent murmur, cannot be accepted as a sufficient cause of a murmur, which, while it lasts, is constant and unchanging. Besides, "there is no good proof that involuntary muscular organs participate in the muscular disorder,"<sup>2</sup> while "the admitted co-existence of rhythmical action of the substance of the heart with this alleged tetanic and irregular contraction of the papillary muscles, which, as proved by dissection, are directly continuous with the fibres of the ventricular walls, constitutes a still stronger objection to this theory,"<sup>3</sup> especially if we "bear in mind the unity of the nerve centres, and the community of nerve-distribution enjoyed by both portions of the same fibres."<sup>4</sup> On the other hand, the wasted skeletal muscles and spanæmic blood of a choreic patient are sufficient indications that similar causes are present in this disease which are known to produce a similar murmur both in chlorosis and in pyrexial diseases, hence there is no difficulty in referring the murmur in chorea to the same causes as in those diseases, nor in recognising that hæmic and dynamic murmurs do not differ from one another but have a similar origin, pursue a similar course, and are amenable to similar treatment.

There is scarcely any form of febrile disease in which so-called hæmic cardiac murmurs have not occasionally been heard. In scarlatina, relapsing and enteric fevers, such murmurs are of common occurrence. In erysipelas, variola,

<sup>1</sup> Walshe, *On Diseases of the Heart and Great Vessels*, 3rd edition (London, 1862), p. 95.

<sup>2</sup> Kirkes, *Medical Times and Gazette* (1863), p. 637.

<sup>3</sup> Hayden, *op. cit.* p. 268.

<sup>4</sup> *Loc. cit.*



measles, and in probably all other febrile diseases, they are occasionally to be heard, but in true typhus such murmurs are practically unknown.<sup>1</sup> When we reflect that the cause of these murmurs is inadequacy of the cardiac contractile force, the result of imperfect metabolism due to flushing with innutritious (spanæmic) blood, we can readily understand that in those whose general metabolism is already impaired a comparatively trifling seizure may give rise to phenomena which, in better nourished patients, are only occasionally observed, even when they have been exposed to a more severe and protracted disease. Hence the want of constancy in the cardiac phenomena referred to in different cases even of the same disease; hence, too, the probably greater frequency of those phenomena in hospital patients than in others. The infrequency of such a murmur in typhus patients is readily understood, when we reflect that to produce sonorous fluid veins at any orifice, a certain amount of energy in the propelling force is requisite, and also remember that the cardiac muscle is so enfeebled in this disease that murmurs due to actual valvular disease disappear during its course, and reappear during convalescence so soon as the heart has re-acquired sufficient force.<sup>2</sup>

In all febrile diseases in which a murmur of the character described is observed, whatever may have been their nature, the sequence of the phenomena, as observed by myself, has been precisely similar to that observed in chlorosis. And it will be readily perceived how important an observation this is; for as these chlorotic and febrile murmurs are actually due to dilatation of the ventricle, and as it is also undoubtedly

<sup>1</sup> For an account of the heart in typhus and in other fevers, I may refer to the classic pages of Stokes in his work *The Diseases of the Heart and Aorta* (Dublin, 1854), p. 366. And an excellent account of the morbid changes induced by protracted fevers in both the voluntary and involuntary muscles is to be found in Zenker's memoir *Ueber die Veränderungen der willkürlichen Muskeln im Typhus abdominalis* (Leipzig, 1864), and in Murchison's work on *The continued Fevers of Great Britain* (London, 1871), p. 248, etc.

<sup>2</sup> Stokes, *op. cit.* p. 444; and recapitulation, § 17, etc.

true that all, or, at all events, all but a small proportion of such cases make a perfect recovery,<sup>1</sup> then we must very considerably enlarge the area of curable cardiac affections, and this we know to be nowadays (1898) duly recognised. In regard to this I do not, of course, refer to the simple vanishing of murmurs due to organic valvular lesions; such lesions are always permanent, as may be readily proved, but the murmurs by which they are ordinarily revealed may, for a time, cease to be heard. In these cases there is no possibility of a cure. It is different in cases of simple dilatation of the heart; in chlorotic and pyrexial dilatation, a perfect cure we know to be the rule; in dilatation from any other cause a cure is perhaps the exception, but it may always be aimed at, and is not infrequently attained. To Senac's mournful wail, "Que peut-on espérer des médicaments, par exemple, dans les dilatations du cœur?"<sup>2</sup> modern medicine can now give a triumphant reply. In simple dilatation of the right heart, the result of bronchitis, the cure of the bronchitis is usually followed by cure of the cardiac affection; but to this there are exceptions, to be subsequently detailed when speaking of affections of the right side of the heart. In dilatation of the left ventricle, occurring at or after middle life and arising from many causes, but having as its basis a weakened myocardium coupled with loss of arterial elasticity,<sup>3</sup> and often precipitated or aggravated by over-indulgence in the pleasures of the table by the well-to-do, or by strenuous exertion accompanied by the free use of alcohol in the poorer classes, treatment is, from the essential nature of the affection, of

<sup>1</sup> Chlorotic murmurs are known ordinarily to result in perfect cure. In regard to murmurs occurring in the course of fevers, Stokes says, "Of many hundreds of cases of weak and softened hearts observed during the last twelve years, we cannot adduce a single instance of organic disease of the heart which could be traced to any injury done pending the typhous affection," *op. cit.* p. 374. And we must remember that under the term "typhous" Stokes included not only typhus, but also enteric and relapsing fevers, *vide* Recapitulation, §§ 37, 38, 39 and 40, *op. cit.* p. 447.

<sup>2</sup> *Traité de la Structure du Cœur, de son Action, et de ses Maladies.* Par Jean Senac (Paris, 1749), vol. ii. chap. iv. p. 328.

<sup>3</sup> *Vide The Senile Heart.*

less avail. Still, even in these cases, marked improvement is the usual result of appropriate treatment, and in a few a positive cure—or, at all events, one of some permanence—is actually attained. The following cases are interesting and instructive examples of this; the cases are much condensed, only the salient points in the cardiac lesion being referred to:—

CASE XVIII. Henry Welsh, a labourer, aged sixty-one, admitted to Ward V., 17th November 1874, complaining of pain in the chest, shortness of breath, swelling of the stomach, and occasional palpitation. About thirty years ago this patient had an attack of rheumatic fever, which he says lasted twenty weeks. For the last ten years he has had occasional pains in the left haunch and arm, which he calls rheumatic. With these exceptions his health was perfect up to last August, when he was suddenly seized with severe pain in the front of the chest, accompanied by shortness of breath and expectoration; this lasted about a week. After recovery from this attack, he found that on attempting to work the slightest exertion produced breathlessness and a great feeling of weakness. The pain in the chest which was felt at first has abated, but left behind a soreness which still continues. About a month after his illness, he first noticed swelling of his feet at night, and this still continues. The patient is a well-built powerful-looking man, nearly six feet in height; his muscles are soft, and he says he has lost much flesh. Till six weeks ago his head had to be raised to alleviate his breathlessness; now he can lie in any posture. Expression of face natural, skin natural, temperature 98·6°. The soreness complained of is referred by the patient to a point about midsternum; it is always present, and, though not very severe, it becomes worse upon any exertion. He has no dyspnoea when at rest, but the slightest exertion produces a choking sensation referred by him to the part where the pain is felt. At first he was much troubled with palpitation, but this is not now so frequent. The pulse is 92, and

is composed of alternate strong and weak beats; at times the force of each beat is fairly uniform, accompanied by intermissions and occasional double beats. The veins generally are not distended, but the external jugulars are slightly so, and pulsate distinctly; they do not, however, fill from below when pressure is made on them by the finger. The cardiac apex beats between the fifth and sixth ribs, four and a half inches from the mesian line; no other pulsation is visible. On palpation the cardiac impulse is felt to be feeble and irregular, both as to time and force. On the left parasternal line, one inch from the sternum, the cardiac dulness does not rise above the third rib. Transversely in the plane of the fourth rib, dulness extends from half an inch to the right of the sternum to four inches to the left of the left edge of that bone. On auscultation in the mitral area, a high-pitched systolic murmur is heard, accompanied in three beats out of four by a partially closed first sound; in the fourth the murmur alone is audible. The maximum intensity of this murmur is in the mitral area, but it is propagated distinctly round to the angle of the left scapula. In the mitral area this murmur is followed by a somewhat obscure second sound. In the tricuspid area a loud but low-pitched systolic murmur is distinctly audible, accompanied by some portion of a first sound, and followed by a second sound, which is sharper than in the mitral area. At the base the first sound is closed; the pulmonary second markedly accentuated. There is no venous murmur. This patient gradually improved under treatment, till, on his discharge on 18th February 1875, the only indication of cardiac disease still remaining was a slight impurity of the first sound in the mitral area, and an almost imperceptible accentuation of the pulmonary second. The tricuspid murmur had entirely disappeared.

Time alone can show whether in this case there was not some slight damage of the mitral valve, dating from his rheumatic attack thirty years ago; or whether, as seems



probable, his lesion was solely dilatation of both ventricles, dating from his febrile attack of last August, which seems probably to have been bronchitic in character. In any case his improvement was both marked and remarkable, and the contraction of both ventricles, but particularly of the right one, unmistakable.

In the following case the nature of the lesion is less open to question, and the improvement—though only temporary—even more remarkable:—

CASE XIX. Euphemia Lawson, a domestic servant, aged thirty-six, admitted into Ward XIII. on 26th September 1872, labouring under breathlessness and swelling of the lower extremities. In this case there was forcible pulsation at the lower part of the sternum, pulsation of the jugular veins, and slight increase of the transverse dulness of the heart. In the mitral area a blowing murmur, and in the tricuspid area a similar murmur of a somewhat lower pitch completely replaced the first sound in the respective areas. These murmurs were propagated into the aortic and pulmonary areas; the aortic second was weakened; the pulmonary second much accentuated. Lawson never had any rheumatism whatever, but she worked hard, and was rather given to the abuse of stimulants. About three months after admission, at the hour of visit, we were suddenly called to her bedside to find her completely unconscious, and paralysed on the left side. The unconsciousness passed off within half an hour; the paralysis of the arm ceased to be noticeable in a few days; and after the lapse of some weeks the paralysis of the leg was only revealed by a slight dragging, which ultimately quite disappeared. Under treatment all her cardiac symptoms also vanished; and at her discharge on 26th March 1873, the only detectable sign of what had originally appeared to be a most serious disease was a slight blunting of the first sound. All the murmurs had quite disappeared, and the accentuation of the pulmonary second could no longer be detected. Lawson felt so well that she left the Infirmary to go direct to service,

and in service she remained for over two years. During that period I had repeated opportunities of examining her heart, and always found it exactly as it was when she was discharged. At last, by a repetition of the original causes, Lawson's heart again dilated, and she was readmitted 19th August 1875, with some œdema of her lower extremities, breathlessness, some rhonchi and frothy, watery sputa, the latter coming on after admission. Her heart was again dilated, with strong pulsation beneath the lower part of the sternum, no perceptible apex beat in the usual position, and all the other signs as already described. The pulsation of the heart was, however, more forcible than it had formerly been; it was obviously more hypertrophied, a condition following of necessity on the primary dilatation. In commenting on the case at that time I stated that I still hoped to be able to restore the heart, but not so perfectly as before, and that slighter disturbances would bring back the dilatation, until at last the case would terminate in the usual manner by increasing dropsy and death from asthenia. There was not a trace of her paralysis remaining, and she improved so much under treatment that she was discharged on 19th September with her heart once more completely rehabilitated. But not for long; as prognosticated, Lawson was not long in coming back to die from asthenia, though not from dropsy. She was readmitted on 9th February 1876, complaining of shortness of breath, pain in the left side, and swelling of her legs. Lawson was two and a half months in the Infirmary, and during that time she suffered much from repeated embolisms, both in the pulmonary and systemic arteries. The history of the case records that she suffered from a perfect "shower" of such plugs. The pulmonary embolisms were twice over the cause of localised pleurisies, which on one occasion raised the temperature to 103°. The clots in the systemic arteries chiefly lodged in the spleen and kidneys, but a few days before her death the brachial artery was occluded at its lower end by an embolus. The temperature

and quantity of urine varied incessantly from these causes. About a month after her admission she suddenly presented that rare form of cardiac irregularity of the bigeminal type, in which the right and left ventricle alternately predominates, the carotid artery and jugular vein pulsating alternately.<sup>1</sup> The ever-recurring embolisms gradually reduced the patient to a state of extreme misery, from which she was relieved by death on 29th April 1876. The Infirmary Pathological Report states:—"Heart enlarged, weighing 1 lb. 2½ oz. Right auricle and ventricle were much dilated, and the tricuspid orifice was enlarged, admitting eight fingers. The auricular appendix was filled with a thrombus which was evidently somewhat old. It was adherent at its periphery to the endocardium, and its centre was partially reduced to a soft yellow pulp by fatty degeneration. The visceral pericardium covering the appendix was white and opaque from chronic inflammatory thickening. The left auricle and ventricle were considerably dilated. The muscular wall of the ventricle was slightly hypertrophied, measuring five-eighths of an inch in thickness. In the left auricular appendix there were several small gray thrombi about the size of peas. In the cavity of the ventricle there were also a number of thrombi, and two of them of large size, being respectively that of a walnut and of a filbert. These were of a dirty gray colour and firm consistence. Superficially they presented a rather delicate reticular arrangement of the fibrin, and on being cut into, the larger of the two was found to present a collection of fatty matter in its centre near one extremity, and a serous cyst near the other. The mitral orifice admitted four fingers; cone diameter 1·55 inch. The mitral and aortic valves presented atheromatous thickening, but were otherwise natural." In the lungs, spleen and kidneys, there were fibroid indurations, recent infarctions, and many cicatrices,

<sup>1</sup> This case, along with another similar one, has been published *in extenso* by the late Professor Roy of Cambridge, who was at that time my resident physician.—*Vide* "On Two Heart Cases which presented a Rare Form of Irregularity," *Edinburgh Medical Journal* (January 1878), p. 594.

the result of embolic processes. This very instructive case teaches us that even after middle life we may occasionally succeed in completely rehabilitating a dilated heart, but to keep it so necessitates a careful avoidance of the original causes of the dilatation, and an equally careful attention to the maintenance of the metabolic equilibrium of the organ. This case presents also a well-marked example of an unusual mode of death, exhaustion due to repeated embolisms, not one of which was in itself serious, but which wore out the patient's strength by their continual recurrence. There was no blood-poisoning, the emboli were perfectly aseptic, and the fatal issue resulted from the continual irritation arising from incessant variations in temperature, accompanied by trifling local inflammations occurring in an organism already exhausted. This case also presented a well-marked instance of that rather rare form of irregularity in which there was not only a bigeminal rhythm of the heart's action, but also a weakening of the alternate pulsations of each heart, so that the jugular vein and the carotid artery were seen to pulsate alternately at the root of the neck. This matter will be referred to subsequently when treating of cardiac irregularity.

But perhaps one of the most remarkable cases of cure of a dilated heart was that of Case XX., A. G., married, aged thirty-three, who had been attacked by typhoid fever twelve weeks before her admission to Ward XIII., early in 1877. In the course of her fever she was prematurely delivered of her sixth child. Considerable flooding took place after the birth of her child, and, as she seemed to be gradually getting weaker, she was sent to the Infirmary. On admission she was intensely spanæmic; her heart was extremely feeble and fluttering; her pulse averaged 150 to 180 per minute, and she seemed to be sinking fast. After a few days of appropriate treatment she regained sufficient strength to be able to turn in bed, and her pulse fell to about 90, and had gained both force and tension. No impulse was to be seen over the cardiac area, but slight venous oscillation was visible over the



jugular veins, and a distinct pulsation was to be felt in the second intercostal space one inch and three-quarters to the left of midsternum.<sup>1</sup> Over the jugular veins a venous hum was audible; over the auricular pulsation a loud systolic murmur was audible, and this was less distinctly heard in both mitral and tricuspid areas as a low, blowing murmur taking the place of the first sound. After some months of treatment she was dismissed cured; all her symptoms and physical signs had entirely disappeared. Her home was not all it ought to have been, and in a few months she returned with a relapse of all her symptoms. She was advised to come into hospital, but as she preferred to remain at home, she was dismissed with a prescription for a ferruginous tonic. She did not improve, however, and she was admitted to Ward XV. in October 1877, labouring under general dropsy, and passing only a small quantity of urine deeply tinged with blood. Her heart, particularly the right side, was greatly dilated; there was a loud systolic murmur in all the cardiac areas, and well-marked jugular pulsation. The essential part of the treatment consisted in the administration of digitalis in large doses for short periods, twenty or thirty minims of the tincture or a proportionate dose of the infusion being given every four hours till a decided effect was produced on the heart. The digitalis was then pretermitted for a time, and during this interval, iron and quinine, or iron and arsenic were freely administered to improve the state of her blood. In this way her strength was gradually improved, her dropsy removed, and her heart contracted, so that in June 1878 she was shown to the Medico-Chirurgical Society without a trace of murmur, without any accentuation of the pulmonary second, and with only a slight increase in the force of the heart-beat (hypertrophy) as the sole indication of the serious condition from which she had recovered.<sup>2</sup> Two

<sup>1</sup> This case as it was when first admitted will be found narrated by my resident, Dr. George Gibson, in the *Edinburgh Medical Journal* (October 1877), p. 302, who has also given a cardiogram of the auricular pulsation.

<sup>2</sup> Vide *Edinburgh Medical Journal* (June 1878), p. 1120.

years subsequently (June 1880) she continued in perfect health, and presented no indication of heart trouble beyond a blunt first sound, and a slight increase in the force of the apex beat.

It might not perhaps be easy to multiply cases quite so remarkable as the one just narrated, but scores of similar cases might be quoted if needful as proof that cardiac dilatation is by no means a necessarily incurable condition. In its earliest stage it may generally be recognised by the presence of a more or less distinct systolic murmur, sometimes accompanied by pulsation in the auricular area; as the affection progresses the systolic murmur passes into the mitral area, and by and by implicates the tricuspid also. In advanced cases there is both mitral and tricuspid regurgitation, and there may be a loud systolic murmur in all the cardiac areas. In its slighter forms cardiac dilatation is readily curable, and is almost invariably cured. Even in its more severe forms it may be much ameliorated, and occasionally cured if the patient be young, but in cases past middle life the conditions are such that we have no right to look for more than amelioration. And yet even amongst them we occasionally stumble on a cure.

In simple chlorotic cases it is sufficient to improve the quality of the blood, and through that the general metabolism, to ensure a perfect cure; but this is not always so easy as it seems. The pale countenance and want of appetite suggest fresh air and sunlight, both most excellent remedies, but of no material avail apart from the one remedy which alone proves efficient in the cure of chlorosis, and that is iron. Of all the various preparations of this drug it does not seem that there is any one that can be specially regarded as of more importance than another in the treatment of chlorosis. Neither is any special dose of more consequence than any special preparation. The point of most importance is to select that preparation of iron which agrees best with the patient, and to give it in the dose and in the manner which

produces least disturbance.<sup>1</sup> Arsenic is often combined with iron in the treatment of such cases, and though it is no longer supposed to have any effect in increasing the number of the red corpuscles or the amount of hæmo-globin, its powerful tonic influence on the heart and lungs, if not also on the blood itself,<sup>2</sup> makes it wellnigh indispensable. As the heart is always more or less affected in all cases of chlorosis, abundance of rest is a most important means of averting more serious cardiac trouble,<sup>3</sup> and a month or more in bed is well known to be a most important adjuvant to other treatment. When cardiac dilatation is more marked the tonic influence of digitalis is of the greatest value, and the dose of the drug as well as the method of administration must vary with the requirements of each individual case. In serious dilatation it is generally well to give large doses of digitalis at regular intervals until a decided effect is produced on the heart, then to premit the drug for a time, giving iron and arsenic during the interval. In less serious cases it is sufficient to give smaller doses of digitalis night and morning, iron and arsenic being given simultaneously along with the food. It is always well to avoid giving iron and digitalis in combination, as sickness is very apt to be thus induced. Of course good nourishing food forms a most necessary part of the treatment, and, when the cure is sufficiently advanced, pure country air, with moderate but gradually increasing exertion. Stimulants are not to be recommended; sometimes they are useful and occasionally necessary, but they must be used with caution as tonics only, and not as stimulants. As an example of the benefit to be derived from tonic doses of digitalis even in apparently the most untoward circumstances, I may mention the case of an old gentleman who first consulted me at the age of seventy-seven on account of attacks of syncope which came on whenever he made the slightest exertion. His heart

<sup>1</sup> Vide "The Treatment of Chlorosis by Iron and some other Drugs," by Ralph Stockman, M.D., F.R.C.P.E., in the *British Medical Journal* for 29th April and 4th May 1893.

<sup>2</sup> *The Senile Heart*, p. 270.

<sup>3</sup> *Op. cit.* p. 47.

was so weak as to be almost imperceptible, his pulse so feeble as scarcely to be felt, and the attempt to walk any distance was sure to be followed by a severe faint. After taking for four years ten minims of tincture of digitalis night and morning, he developed a firm forcible apex beat, and an excellent pulse, and he also ceased to alarm his friends by fainting. In short, instead of dying from a so-called fatty heart, as had been predicted, he lived to be over ninety years of age, and finally did not die from his heart at all but from senile asthenia.



## LECTURE VII

### ON TRICUSPID REGURGITATION, CURABLE AND INCURABLE

IN speaking of the murmurs connected with stenosis of the mitral opening, I referred to one case in which stenosis of the tricuspid was not only suspected during life, but actually proved to exist by the results of a *post-mortem* examination.<sup>1</sup> Since then we have had another case, in Ward V., in which the peculiar propagation of the presystolic murmur left no doubt on my mind that we had before us another example of stenosis of the tricuspid as well as of the mitral opening. Happily we were able to discharge this patient considerably improved, and are therefore unable to speak with perfect certainty as to his actual lesion.<sup>2</sup> The morbid appearances found in two other cases of tricuspid stenosis have also been recorded by Dr. Haldane; in one of these the diagnosis was made during life.<sup>3</sup> Thus in little more than ten years there have been four cases of tricuspid stenosis observed in the Edinburgh Infirmary, so that, though an infrequent affection, we cannot afford altogether to ignore it. Tricuspid stenosis, when not congenital, is very generally—79 times out of 117 cases—associated with stenosis of the

<sup>1</sup> *Vide* Case IX. p. 122.

<sup>2</sup> Case of Martin Kelly, aged sixteen, admitted to Bed 6, Ward V., on 26th January 1874; discharged 19th March *eodem anno*.

<sup>3</sup> *Edinburgh Medical Journal* (September 1864), p. 271. These are all the marked cases of tricuspid stenosis that occurred during the ten years specified, but I am quite conscious of having during that time occasionally seen in the *post-mortem* theatre other hearts in which the segments of the tricuspid valve were thickened and slightly shortened.

mitral opening also, and is mostly of rheumatic origin.<sup>1</sup> Tricuspid stenosis has in every case a most unfavourable influence upon the prognosis, and is always a most serious complication of mitral stenosis. It is scarcely possible to make an accurate diagnosis of such a condition; the points to be mainly relied upon are, first, the presence of a presystolic murmur or thrill, or of both of these signs, in the tricuspid area and extending to the right of the sternum. Unfortunately both of these signs are often absent. The second important indication never fails, only *variat gradu*, and consists in the evidence of considerable backward pressure, with but little indication of regurgitation; well-marked dulness to the right of the sternum, in the plane of the fourth rib—the auricular area—accompanied by considerable turgescence of the jugular veins without pulsation, or with only an inconsiderable indication of pulsation. Naturally this sign only affords presumption, and is not infallible. Mitral stenosis in its most usual form involves regurgitation also, and the same is true of tricuspid stenosis, as has been frequently recognised.<sup>2</sup> Regurgitation occurring in such a manner is absolutely incurable. I have already mentioned that pure tricuspid regurgitation—the result of simple dilatation—is a frequent concomitant of mitral stenosis, and that it occurs sooner in such cases, and is more considerable, the earlier in life the mitral stenosis has been developed, especially if it is associated with any marked degree of aortic hypoplasia, being almost invariably in all such cases the mere prelude to the end.<sup>3</sup> In cases such as these the tricuspid regurgitation is always considerable, and its effects upon the systemic venous circulation well marked. It is of course incurable, though, as already noted, it may be—especially at first—somewhat modified by treatment, and the inevitable

<sup>1</sup> Vide *Essai sur le Rétrécissement Tricuspidien*, Thèse pour le Doctorat en Médecine par M. Robert Leudet, Paris, Steinheil, 1888.

<sup>2</sup> *Edinburgh Medical Journal* (September 1864), pp. 271, 272; and Leudet, *op. cit.*

<sup>3</sup> Lecture V. p. 135.

end postponed. The amount of relief to be thus obtained depends upon the degree of stenosis present, as well as on that of the aortic hypoplasia. But the dilated condition of the right ventricle, though for a time it may be remedied, speedily becomes persistent, and the hypertrophy ere long becomes considerable, so that at times the right ventricle forms the apex of the heart, and the left ventricle seems a mere appendix attached to it.<sup>1</sup>

The physical signs upon which we rely as certain indications of tricuspid regurgitation are few, simple, and easily recognised. First, there is a shifting of the apex beat from its normal position, about two inches to the left of the sternum in the fifth interspace, to just beneath the sternum, and also a change in its character from an outward thrust to a diffuse impulse more or less heaving and forcible in proportion to the hypertrophy present. These variations from the normal result from the dilatation of the right ventricle and arise from its anatomical position in front of the left. Similar modification may be temporarily brought about even in health by holding the breath as long as possible ; respiratory changes being thus stopped, the lungs become congested, the right ventricle, dilated by residual accumulation, swells out like a water cushion in front of the left ventricle, which it pushes backward into the cavity of the chest ; the apex fails to reach the chest wall and ceases to be felt by a finger resting on it, while the palm of the hand placed over the lower part of the sternum feels an obscure pulsation. This is an experiment which each one can make for himself.

Second, on percussion there is increased dulness to the right of the sternum in the plane of the fourth rib ; this, however, is rather the result of secondary dilatation of the right auricle than of any increase in the breadth of the ventricle.

Third, on auscultation the stethoscope in the very earliest

<sup>1</sup> Case XIV. p. 143. Many similar cases have been observed ; those recorded are selected representative cases, and are to be accepted as typical examples of a group, and not merely as isolated instances of any given abnormality.

stage of dilatation of the right ventricle conveys to the ear a feeling of unusual and abnormal pulsation in the tricuspid area. Later on, in the same area, viz. at the sternal end of the fourth, fifth, and sixth ribs, we may have a loud systolic murmur, which is propagated with more or less distinctness into all the other cardiac areas, but which has its position of maximum intensity in the position referred to. When mitral co-exists with tricuspid regurgitation we have two foci of maximum intensity for the systolic murmur, one in the tricuspid, and another in the mitral area or a little to the left of it; there is often also a difference of pitch in the two murmurs, but sometimes the one and sometimes the other is the higher. Like all cardiac murmurs the tricuspid is occasionally absent, even when all the other signs of tricuspid regurgitation are certainly present, a murmur being always the least reliable sign.

Fourth, even without any notable regurgitation through the tricuspid valve, long-continued pulmonary congestion and overfilling of the right ventricle are necessarily followed by great congestion of the right auricle and of the systemic veins. Hence in such cases we have the external jugular veins, and especially the right one, markedly turgid, and frequently undulating with a wavy motion communicated by the systolic percussion of the turgid tricuspid valve acting through the medium of the blood in the overdistended right auricle, which has all the elasticity of compressed fluid. But when long-continued distention, due to tricuspid regurgitation, has made inefficient the valve at the root of the external jugular where it enters the subclavian, we have distinct pulsation in the jugular vein, the vessel filling from below synchronous with each ventricular systole. This venous pulsation is always more or less visible, according as the veins are more or less distinct, but is best marked in elderly persons, in whom the veins are usually larger, and the adipose tissue covering them not so abundant as in younger people. By compressing the vein at the middle of the neck, we stop



the downward flow from the head and the systolic reflux is more easily seen. Jugular pulsation is generally visible whenever we have a tricuspid murmur, and it is sometimes to be seen when no murmur is to be heard. As it is always a result of great previous venous turgescence, it may be accepted as a reliable indication of tricuspid regurgitation—even though a murmur be absent—provided the other signs just referred to are present. But if the substernal pulsation be absent or slight, it is possible that the jugular pulsation may only be the remanent result of a pre-existing tricuspid regurgitation, the valve being perfectly rehabilitated—incompetence of the venous valve causing what would otherwise have been a mere undulation to exhibit a distinctly pulsatory character. Jugular pulsation is thus always either a proof of existing tricuspid regurgitation, or a sign that tricuspid regurgitation has previously existed, and may be readily reproduced. It is therefore a sign of some importance. Pulsatory and other abnormal changes are most readily observed in the jugular vein, but the jugular is by no means the only vein affected by a systolic reflux. From the absence of valves, the considerable size, and the dependent position of the *vena cava inferior*, the regurgitant blood must flow with great freedom and directness down that vessel into the liver. Hence hepatic pulsation is a phenomenon that may be observed, in many cases, even before pulsation can be seen in the jugular vein. It is well to remember, however, that hepatic pulsation may be produced in three different ways. First, we may have a simple depression of the liver, chiefly the left lobe, synchronous with the systole of the ventricles, and apt to be regarded as a pulsation *in scrobiculo cordis* of the heart itself. Second, we may have a heaving of the whole mass of the liver, due to a movement communicated to it by an aneurysm of the descending aorta, or by the *vena cava*, which may be enormously dilated.<sup>1</sup> This pulsation is not synchronous with the ventricular systole, as

<sup>1</sup> *Vide antea*, p. 8.

may be readily demonstrated by the measures formerly described (p. 9). And, third, we may have a distinctly expansile pulsatory movement also communicated to the whole mass of the liver by the systolic pulsation of the hepatic veins ramifying within it. This pulsation is also delayed and is not synchronous with the ventricular systole; its expansile character sufficiently distinguishes it from that form of hepatic pulsation most nearly allied, which has just been described. Excluding the pulsation produced by aneurysm, the two latter forms of hepatic pulsation are indications of a more advanced and more serious degree of cardiac dilatation than suffices to produce the variety first described. Hepatic pulsation which is retarded and is not synchronous with the cardiac systole is, therefore, an important diagnostic phenomenon, and every care must be taken to expiscate its ultimate cause. There is too much reason to suppose that these varieties of hepatic pulsation have not always been sufficiently discriminated, and that hepatic pulsation has been, occasionally at least, simply referred to the ventricular impulse, even when the absence of synchronism has been distinctly recognised. Allan Burns, for example, refers to a most furious pulsation in the epigastric region, "produced," as he says, "entirely by the dilated heart," in which, from the absence of synchronism between the heart and the pulsating liver, no one ever doubted but that the cause of the pulsation was an aneurysm of the coeliac artery.<sup>1</sup> In very rare cases we may have as the result of tricuspid regurgitation a reflux pulsation visible not only in the jugular, but also in some of the smaller and more superficial veins—as the brachial—but this is so uncommon as to be unimportant.

It is commonly said that tricuspid regurgitation alters or removes any pre-existing accentuation of the pulmonary second sound. No doubt it must alter this accentuation, but inasmuch as we have not the two conditions simultaneously

<sup>1</sup> *Op. cit.* p. 264.

before us, we never can say to what extent or in what manner it is changed. But tricuspid regurgitation can never altogether remove pulmonary accentuation, because the same increased intra-pulmonary blood-pressure which produces and maintains the regurgitation also keeps up the accentuation. So long, therefore, as tricuspid regurgitation persists the pulmonary second must continue to be accentuated relative to its norm, and must be markedly so in relation to the aortic second, which from the same cause is weakened.

As can be readily understood, and has been already pointed out, mitral stenosis is a frequent cause of serious tricuspid regurgitation, and the earlier in life the stenosis has occurred the more speedily, as a rule, does the tricuspid regurgitation follow, and the more serious is the prognosis. But any hindrance to the circulation through the left heart may be a cause of tricuspid regurgitation, hence simple mitral regurgitation, due to dilatation only, is not infrequently associated with this affection, but this is not common in early life, is never relatively so excessive, and the dilatation and hypertrophy usually progress on both sides *pari passu*. At first by treatment we may be able to rehabilitate the tricuspid valve, but unless we can also rehabilitate the mitral we only gain a temporary postponement of the end. Still this end is never so rapidly reached as in serious mitral stenosis. And from the simultaneous implication of both ventricles, and the long duration of the disease in many such cases, the whole heart is often enlarged to a veritable *cor bovinum*,<sup>1</sup> before the gradual transference of the blood from the arteries to the veins culminates first in general dropsy, and lastly in fatal asthenia, and skilful treatment may do much to mitigate the evils of this condition and to postpone the inevitable end. Great obstruction at the aortic orifice has a similar effect to mitral stenosis, but extreme aortic stenosis is so rare as to be of little practical

<sup>1</sup> Theodor. Priscian, *De Diaeta*, 15, speaks of "Medulla bovina." Though unusual, the adjective is strictly classical.

importance as a cause of tricuspid regurgitation. On the other hand, aortic regurgitation is common enough, but its own peculiar sources of mortality are such as only too frequently anticipate any important alteration of the right ventricle.<sup>1</sup> It is only comparatively rarely, therefore, that aortic incompetence gives rise to tricuspid regurgitation of a serious character.

Next to diseases of the left heart, diseases of the lungs are those which affect the right ventricle, and only two of those are of much importance in this respect. Chronic, and especially tubercular diseases of the lungs only exceptionally produce any alteration of the right ventricle, because with the progress of these diseases there is a simultaneous diminution of the amount of blood circulating. Such acute diseases as pneumonia never last long enough to produce any serious alteration of the right heart; and even pleurisy with considerable effusion is seldom sufficiently protracted without so much diminution of the mass of blood as to make consecutive dilatation of the right ventricle a comparatively rare complication. It is otherwise with bronchitis—acute or chronic. In it the obstruction to the circulation through the lungs is great, lasts sufficiently long, and is accompanied by no diminution in the mass of blood sufficient to nullify its dilating effect upon the right ventricle. In bronchitis, therefore, we have, next to mitral stenosis, the most common cause of considerable tricuspid regurgitation. But inasmuch as bronchitis is a curable disease, the tricuspid regurgitation produced by it is evanescent, passes away with its cause, and does not always recur, even with a recrudescence of the exciting and primary disease; because to produce cardiac dilatation there must be, in addition to bronchitis, some loss of tone of the cardiac muscle due to pyrexia or defective metabolism, the result of impaired nutrition either of general or local origin.

Emphysema of the lungs, which is the only other pulmonary cause of tricuspid regurgitation of any importance, is a much more serious cause than bronchitis, because from

<sup>1</sup> Lecture III. p. 95.



what we know of its pathology the presence of emphysema necessarily presupposes a greater or less limitation of the area of the pulmonary capillaries, and consequently an obstruction to the circulation through the lungs as permanent and as incurable as that arising from any equivalent amount of mitral stenosis. Tricuspid regurgitation following emphysema of the lungs has, therefore, quite as serious a prognosis in regard to curability as that following mitral stenosis, but, as we shall presently see, the prognosis *quoad* longevity is not by any means so grave. Tricuspid regurgitation from emphysema has, however, certain physical signs peculiar to itself, to which I shall presently refer.

The following case presents a well-marked example of curable tricuspid regurgitation:—

CASE XXI. William Savage, aged thirty-eight, a labourer, admitted to Bed 3, Ward V., on 24th March 1870, complaining of great difficulty of breathing, and of cough and expectoration. On admission the patient stated that he worked in a quarry, and frequently suffered from chills, the result of exposure to cold winds while heated by hard labour. The present attack was the result of one of those chills. He had repeatedly suffered from similar attacks, and in two similar seizures he had been treated by copious and repeated venesections. The patient was tall and strongly built. His countenance was swollen and dusky; he suffered from orthopnoea; his skin was cool, and his finger-tips purple. On inspection his apex beat could not be seen, but instead there was a pulsation beneath the lower part of the sternum and in the *scrobiculus cordis*. On palpation the chest was felt to expand equally in all directions, and a systolic heaving impulse was felt beneath the lower part of the sternum, the apex beat being absent from its normal position. The percussion note over the chest was normal, except a slight increase of the transverse dulness of the heart, which, at the level of the fourth rib, commenced nearly one inch to the right of the sternum. On auscultation moist crepitations,

mingled with a few rhonchi, completely obscured all other pulmonary and cardiac sounds, except that the pulmonary second was distinctly heard to be accentuated. A large jacket-poultice of linseed meal was ordered to be applied over the whole chest, and half an ounce of infusion of senega with ammonia and spirit of chloroform was given every four hours. *25th March.*—Had slept badly on account of his orthopnoea. The whole surface was livid; condition otherwise unchanged. Infusion of digitalis to be substituted for that of senega. *26th March.*—To-day there was evident improvement, and a systolic murmur could now be heard in the mitral area, replacing the first sound. A systolic murmur could also be heard in the tricuspid area, and in the aortic area the first sound was replaced by a murmur, the semilunar valves being distinctly heard to close. Next day, *27th March*, the lungs were so far free from crepitations that it was possible accurately to differentiate the mitral from the tricuspid murmur, and the patient could now sleep well in the recumbent posture. On *29th March* the patient felt almost well, and had but little cough. His breathing was easier, and his colour natural. On *1st April* it was noted that though the tricuspid murmur was still distinct, yet the pulsation of the right ventricle was mostly confined to the lower part of the sternum, and was hardly perceptible in the epigastric region. On *2nd April* it was noted that the chest was quite free from crepitations, and that no murmur was to be heard in any of the cardiac areas. On *9th April* he had an attack of pleurisy on the right side accompanied by friction, but this was unattended by any return of the cardiac symptoms. Under the use of poultices and iodide of potassium this attack was recovered from in two or three days, and the patient was discharged well on *18th April*. On *15th December* of the same year (1870) this patient returned to hospital, complaining of dyspnoea, cough, pain over the lower part of the sternum, and slight pulsation in the epigastrium. On auscultation the cardiac sounds were

found to be distinct and free from murmur, the pulmonary second accentuated. Fine crepitation was audible over the right base anteriorly; percussion everywhere normal. A jacket poultice and rest in bed were prescribed, and on 26th December he was discharged perfectly well.

The first remark I have to make in regard to this case is that, recognising the improbability of a mitral murmur co-existing with a tricuspid murmur of regurgitation, both apparently due to pulmonary obstruction, every pains were taken to make sure of our facts. And the co-existence of these two murmurs in such a case must, I think, be accepted as an important proof of the influence of pyrexial relaxation in producing cardiac dilatation, even in circumstances otherwise unfavourable for such a development.<sup>1</sup> If by chance we were mistaken in our assumption of the presence of a murmur of mitral regurgitation in this case, this in no respect invalidates the importance of the case as a remarkable instance of curable tricuspid regurgitation—of which all the other signs, including venous pulsation, were present—depending upon pulmonary obstruction of a temporary character. I have selected this case as an example of this affection mainly because we had in him a subsequent opportunity of observing the permanence of the cure. The following case is an equally instructive example of incurable tricuspid regurgitation, incurable not because the right ventricle was dilated, but because that dilatation was

<sup>1</sup> Left-side dilatation is a common enough accompaniment of right-side dilatation. The singularity in the above case consists in the dilatation of both sides being associated with and apparently dependent on the bronchitis. Evidently the dilatation of the left heart had been primary and due to pyrexia, the dilatation of the right heart being secondary, and due to pulmonary congestion and the damming back of the blood. It is curious that C. Gerhard, who distinctly recognises the influence of pulmonary emphysema in producing dilatation and hypertrophy of the right heart, should yet refer the systolic murmur accompanying this to *mitral* regurgitation. He calls this so-called mitral systolic murmur “ein Zeichen localer Anaemie,” and supposes it to be produced in some inexplicable manner by a defective blood supply to the left side of the heart and the arteries.—Vide *Lehrbuch der Auscultation und Percussion* (Tübingen, 1871), S. 264; vide also Lecture VI.

produced by a permanent and irremediable form of pulmonary obstruction.

CASE XXII.<sup>1</sup> Andrew Laidlaw, a cabinetmaker, aged forty-nine, was admitted into Bed 2, Ward V., on 23rd October 1874, complaining of indigestion and breathlessness. He stated that about two years ago he first suffered from shortness of breath while at work, accompanied by a feeling of oppression in his chest, a troublesome cough, spit, and severe pain darting through the chest, from before backwards, on the occurrence of any severe paroxysm of coughing. Mustard poultices had been successfully employed for the relief of this pain. He also stated that he had suffered from rheumatic fever, for which he could give no date; and that, at the age of nineteen, he had an attack of measles, and that he has never since been free from cough and spit. His personal and family history are unimportant; he has always had a comfortable home, and from the nature of his occupation he has not been much exposed to atmospheric vicissitudes. The patient is a man of fair muscular development, 5 feet 7 inches in height; his expression is natural. He suffers from dyspnoea whenever he makes any exertion. He has a good deal of cough, which causes him some pain, and is accompanied by the expectoration of about five ounces of muco-purulent sputa daily. Two years ago he had slight hæmoptysis. His respirations are 30 per minute. His pulse is 80, soft and full, and he occasionally suffers from palpitation and pain at the præcordia. On inspection his thorax seemed emaciated, and the intercostal spaces were apparently distended, giving a rounded barrel-shaped appearance to the chest. The rhythmical movements of the thoracic walls seemed somewhat restricted, and were unbroken by tapping of the heart's apex; but instead a distinct pulsation was seen in the epigastric region, and from this a pulsatory tremor passed over the region of the liver. On palpation

<sup>1</sup> Reported by Mr. C. S. Roy, clinical clerk.



both sides of the chest were found to expand equally but imperfectly, especially over the upper part, and on passing a tape round the chest, in the plane of the fourth rib, the difference between deep expiration and full inspiration was found to be only half an inch. No cardiac impulse was to be felt in any part of the præcordia, but a distinct limited pulsation was felt in the left side of the epigastric region, and synchronous with this there was also felt an equally distinct but somewhat tremulous pulsation which extended over what was subsequently ascertained to be the area occupied by the liver. The percussion note anteriorly was hyper-resonant over both lungs, especially above the fourth rib. On the right side in the nipple line this hyper-resonance continued to the upper border of the fifth rib; beneath that the liver dulness extended downwards for a distance of five inches. On the left side, one inch from the sternum, the percussion note was hyper-resonant down to the third interspace; from the upper border of the fourth rib the percussion note gradually rose in pitch and shortened in duration down to the level of the sixth rib; below this the tympanitic sound of the stomach alone was heard. On percussing across the chest at the level of the fourth rib, cardiac dulness was noted as commencing at the right edge of the sternum, and it extended across to the left nipple, a distance of four inches. Posteriorly, the percussion note was not only lower in pitch and of longer duration than usual (hyper-resonant), but it could also be carried down to below the twelfth dorsal vertebra, though on the right side the percussion note began to rise in pitch and to shorten in duration directly the upper border of the sixth rib was passed. On auscultation over the lungs the respiration was everywhere heard to be indeterminate, presenting the character of an imperfect inspiratory vesicular murmur, followed by prolonged expiration, accompanied by sibilant rhonchi. On listening in the normal cardiac areas the heart sounds were

heard faintly, but free from murmur. At the base of the heart the aortic and pulmonary second sounds were heard more distinctly at the sternal ends of the third rib on the right side, and of the fourth rib on the left side, than in their normal position at the sternal end of the second right and third left ribs. Over the pulsation in the epigastrium a soft blowing murmur was heard occupying the time of the first sound. The patient's tongue was large, pale, flabby, and indented by the teeth, which were much decayed. His appetite was indifferent, deglutition perfect, but he occasionally vomited the food he took. On palpation and percussion the liver was ascertained to come down below the ribs, and it also lay below the pulsating tumour in the epigastrium. The splenic dulness was not enlarged. The urine was of a dark sherry colour, specific gravity 1022, acid reaction, deposited a slight mucous cloud on standing, and contained nothing abnormal. He suffered occasionally from severe frontal headache, had sometimes been deaf, and since shortly after the commencement of his last illness he had been obliged to use spectacles, but he had no other symptoms of nervous change. After about a week's treatment, consisting of five grains of carbonate of ammonia in half an ounce of infusion of digitalis every four hours, an impure first sound was occasionally—about every third beat—to be heard over the epigastric pulsation. But the right ventricle never became more perfectly rehabilitated, and though the bronchitis speedily disappeared, no treatment had any further effect on the condition of the heart. On 4th February 1875 he was discharged free from bronchitic complication, but otherwise in *statu quo*.

This very interesting case was sent to the Infirmary supposed to be labouring under aneurysm of the abdominal aorta. But an aneurysm of the aorta reaching the surface in the position of the pulsating tumour in this case, just below the ribs in the epigastric region, has its pulsation always more distinctly expansile in character, and the

pulsation itself, as well as any murmur that may be present, are always delayed beyond the systolic pulsation and sound of the heart, which in this case they were not. The conclusion we arrived at was that the right ventricle had been depressed below its normal position, that it was dilated, and that the tricuspid valve was incompetent. The physical signs already detailed not only agree in confirming this, but they also teach us that the cause of this dilatation of the right ventricle is of such a nature as to render it permanently incurable.

The resonant percussion in the præcordial area is not of itself sufficient to determine depression of the heart; nor is the co-existence of a pulsating tumour in the epigastrium necessarily any aid in establishing such a diagnosis; because emphysematous hyper-resonance of the left lung might extinguish cardiac dulness without depressing the heart; while epigastric pulsation, even if it were clearly ascertained to be cardiac, and not merely hepatic, might be solely due to dilatation of the right ventricle. But when we find that the vertical cardiac dulness does not commence till a whole rib and an interspace below the normal position; when we also find that this vertical dulness carried down in the usual parasternal line rests for rather more than a similar space upon a tumour whose pulsation is the only movement to be felt in that region, and that this pulsation is synchronous with a sound that occupies the appropriate rhythmical relation of a first sound to the aortic and pulmonary second sounds, which, moreover, have their position of maximum intensity depressed in exact relative proportion to the apparent depression of the entire cardiac dulness, the conclusion seems irresistible that we have to do with an actual and not merely an apparent depression of the heart. And this conviction gains strength when we find that notwithstanding considerable enlargement of the liver that organ is also displaced downwards, and especially when we find that the condition of the lungs is such as efficiently to account for this downward displacement of

both organs. The hyper-resonant percussion note indicates the presence of an abnormal amount of air in the lungs, especially in their upper lobes; while the defective expansion of the chest, the imperfect character of the inspiratory murmur, and the prolongation of the expiration, all indicate that this abnormal condition depends upon retention of air within the pulmonary tissue. This retention of air within the lungs we know happens as the result of that loss of elasticity always associated with over-distention and rupture of the air-cells distinctive of emphysema, which is always accompanied by inelastic enlargement of the parts of the lung affected. This condition, if sufficiently extensive, is invariably associated with depression of the heart and liver, and with extension downwards—as in this case—of the area of pulmonary resonance. Further, as the ultimate branches of the pulmonary artery are distributed on the walls of the air-cells, any destruction of these walls must be accompanied by a corresponding limitation of the area of the pulmonary capillaries. Consequently we have in all cases of extensive emphysema an irremediable obstruction to the circulation through the lungs, which must infallibly produce an incurable dilatation of the right ventricle, with tricuspid regurgitation. The physical signs in this case give thus a perfectly sufficient explanation of all the phenomena present, upon the only tenable supposition, viz. that we have to do with a case of emphysema of both lungs, producing depression of both heart and liver, dilatation of the right ventricle, regurgitation through the tricuspid valve, and secondary enlargement of the liver from venous engorgement. It cannot be denied that a history of thirty years of more or less constant bronchitis is quite sufficient to account for the emphysema present, into the pathology of which we shall not enter further at present.

This case is one of the best-marked examples of incurable tricuspid regurgitation I have ever seen, and with it I conclude the history of this lesion.



Careful attention to all the phenomena described will enable any one readily enough to detect enlargement of the right ventricle, and to determine its degree and its seriousness as measured by that. In this respect you will observe that in the case last narrated, though from its cause it was certainly incurable, yet the regurgitation was not great. This we deduce from several facts: first, the right ventricle could be partially rehabilitated; after but a short time of treatment an impure first sound could be heard every third beat or so. And, second, there was no evidence of any great venous remora; there was no venous pulsation in the neck; there was no true or distensile pulsation of the liver; the pulsation of the liver was synchronous with that of the heart, and was not delayed (p. 201); there was never any trace of albumin in the urine; and there was never any dropsy. The prognosis in this case is not absolutely unfavourable; the tricuspid regurgitation is certainly incurable, but with care he may live a long time. His cardiac affection will assuredly prove fatal in the long run, but in itself it may take a long time, and he is much more likely to be cut off by some intercurrent pulmonary attack.

In regard to prognosis, you will remember that tricuspid regurgitation associated with, and wholly caused by an acute attack of bronchitis, is a perfectly remediable affection, and is often permanently cured. It is a measure of the degree of pulmonary congestion present, and to that extent of the serious character of the pulmonary disease, but in itself it is of little consequence.

But when associated with mitral disease, and especially with mitral stenosis, as it so often is, the prognosis of tricuspid regurgitation is always most grave. Under these conditions, even when dilatation of the right side has been precipitated by an acute attack of bronchitis, its rehabilitation is extremely difficult, and it is never more than temporary; the end is always hastened, and is often rapid in its advent.

In connection with pulmonary emphysema tricuspid regurgitation is equally incurable, but the prognosis as to life is not so grave. It is not difficult to understand why this should be, for we know that the pernicious influence of tricuspid regurgitation is not to be estimated by the degree of venous stasis produced, but rather by the diminution of aortic blood-pressure to which it gives rise, of which the venous stasis is only an approximate measure. A limitation of the area of the pulmonary capillaries, though it may suffice to induce tricuspid regurgitation, diminishes the amount of blood in the aortic system only by the quantity of blood actually cut off. So long as this is consistent with the maintenance of life, no important change can take place without the intervention of secondary causes. Nay, an improvement in the patient's condition is more likely to occur than the reverse, because improvement must necessarily follow any rehabilitation of the right ventricle, or any increase of function in the healthy parts of the lung, both of which are certain to follow improvement in the patient's health, the result of successful treatment. Hence tricuspid regurgitation arising from pulmonary causes compatible with life may, though incurable, persist indefinitely until the aortic blood-pressure becomes fatally lowered by some secondary cause.

But the supervention of tricuspid regurgitation upon mitral stenosis has quite another and a more important signification. Physical laws teach us that the force and velocity with which any fluid passes through an orifice are increased in a certain definite ratio by the amount of pressure brought to bear upon it. It is evident, therefore, that the amount of blood passing through a stenosed mitral orifice during the ventricular diastole considerably depends upon the amount of hydrodynamic pressure within the pulmonary circuit. And it is equally evident that whenever the intrapulmonary blood-pressure is lowered by any efficient cause, such as tricuspid regurgitation, the immediate result is a diminution of the flow through the stenosed orifice, and a

corresponding lessening of the amount of blood thrown into the aorta. Further, the amount of blood sent forward is diminished with a rapidity and in a degree commensurate with the freedom of regurgitation and with the influence which the pre-existing hydrodynamic pressure has had in maintaining the flow; further, this reduction in quantity has a strong tendency to be permanent, and to increase at a rate proportionate to the degree of stenosis present, and with the amount of tricuspid regurgitation.

In tricuspid regurgitation depending upon pulmonary obstruction, therefore, the diminution in the amount of blood sent forward, and the consequent impairment of all the secretions and other vital functions, is to be estimated merely by the amount of blood thrown back. But when tricuspid regurgitation depends primarily upon mitral stenosis, its influence in lessening the amount of blood thrown into the aorta is to be measured not by the ratio between the stenosed and the normal mitral opening, but by that plus the lowering of the hydrodynamic pressure within the pulmonary circuit which the regurgitation has brought about. There is no difficulty, therefore, in understanding which is the most serious lesion, nor why it should be so; even if both are equally incurable. In tricuspid regurgitation resulting from emphysema alone, the patient may continue in moderate health for long, though he will never be robust, and will always be placed in extreme danger by any attack of acute pulmonary congestion. But in that resulting from mitral stenosis, the case is far otherwise.

In the treatment of tricuspid regurgitation, whatever subsidiary measures may be employed, we must never omit the use of digitalis, a drug which, by improving the elasticity of the myocardium, not only benefits its metabolism but also promotes the rehabilitation of the tricuspid valve.

Even in tricuspid obstruction, of which I have seen but little, there appears to be no reason for withholding digitalis. In both of the two cases we had under treatment,

that drug did good service. And we can readily understand that, as its action is not limited to the ventricles, but is exerted on the auricles also, digitalis must do good service in every case in which improvement in the elasticity and of the metabolism of the myocardium is likely to be of service. Nay, more, as digitalis acts upon all muscular fibres in a similar manner, the improved elasticity and metabolism of the muscular fibres of the circulatory system generally, both arteries and veins, cannot but have an important effect in increasing the energy of that systolic wave that passes over the whole circulatory system, upon which the maintenance of the circulation in many of the lower animals so entirely depends, and which is certainly not without an important influence in maintaining the circulation even in the highest mammals.<sup>1</sup>

<sup>1</sup> Note on independent pulsation of the pulmonary veins and vena cava, by T. Lauder Brunton, M.D., F.R.S., and Sir J. Fayrer, M.D., K.C.S.I., *Proceedings of the Royal Society of London*, vol. xxv. No. 172, p. 174.



## LECTURE VIII

### ON THE MURMURS AUDIBLE IN THE PULMONARY AREA, THEIR VARIETIES, AND DIAGNOSTIC SIGNIFICATION

THE pulmonary area, the space between the second and third ribs just to the left of the sternum,<sup>1</sup> has been not inaptly termed the region of romance, on account of the variety of interpretations given to the murmurs having their position of maximum intensity in that position. These interpretations have been only too apt to vary with the views and predilections of the commentators; and yet there is really no place for opinion in the interpretation of a murmur, whatever its position, as that interpretation must rest upon those laws upon which physical diagnosis is founded. The correctness, therefore, of any diagnosis depends far more upon the skill of the observer in collecting the necessary facts, and his ability to draw the legitimate conclusions from those facts, than upon any mere ripeness of experience. The readiness and accuracy with which we can now collect from several points of view the various facts which reveal the physical condition of the heart, and especially of its orifices, leave but little room for the influence of mere opinion on the diagnosis of diseases of the heart.

Systolic murmurs, duly recognised as such by their synchronism with the beat of the heart's apex or of the carotid artery, and most distinctly audible between the second and third ribs to the left of the sternum, have two foci of maximum intensity. In one series of such murmurs the position of maximum intensity is not close to the sternum,

<sup>1</sup> *Vide* Lecture I. p. 29.

though the murmur is also distinctly audible there, but half an inch or more to the left of that bone (*vide* p. 171), just where we know that in most cases (Walshe) the appendix of the left auricle comes up from behind to the left of the pulmonary artery (*vide* p. 170). When much dilated the auricle may extend still farther to the left as well as downwards, and occasionally partly covers the pulmonary artery. Yet even in its most dilated condition there is always a space between the stethoscope resting on the position of maximum intensity of the murmur and the left edge of the sternum equal to at least the breadth of the tip of the middle finger. A murmur of this character, though often attributed to the pulmonary artery, is really formed in the left auricle and depends upon regurgitation through the mitral orifice, as was first pointed out by Naunyn.<sup>1</sup> A systolic murmur in this position is a very common one, and in by far the greater number of cases it betokens merely a simple and curable dilatation of the left ventricle, of which it is an early sign. In chlorosis and allied conditions it is associated with a venous hum and with other signs and symptoms of spanæmia, and it commonly passes for a hæmic murmur. I have already told you my reasons for adopting Naunyn's interpretation of this murmur, and have pointed out the means of diagnosing these cases and of curing them.<sup>2</sup> In a smaller proportion of cases, but still sufficiently common, a systolic murmur with its position of maximum intensity in this position is due to regurgitation through the stenosed mitral opening, conditions which almost<sup>3</sup> always co-exist. Many circumstances favour in these cases the convection of the murmur upwards to the auricular, rather than its conduction downwards into the mitral area. For instance, the tip of the

<sup>1</sup> Loc. cit., *Berliner klinische Wochenschrift* (1868), No. 17, S. 189.

<sup>2</sup> Lecture VI., *passim*.

<sup>3</sup> I have employed the word "almost" in this connection, because there are a few funnel-shaped valves in which it seems impossible for regurgitation to take place at all, and in which at all events it can only take place with difficulty and to a trifling extent.

left auricular appendix lies normally in this position (*vide* frontispiece, also p. 164), and the appendix tends, when dilated, to come to the front round the base of the heart. Next there is the readiness with which the slightest dilatation of the right ventricle pushes the left apex backwards into the thoracic cavity and away from the chest wall (*vide* p. 169). And, lastly, there is the natural tendency of murmurs to be propagated to the surface through the organ in which they are produced (in this case by impingement of regurgitant fluid veins upon the tense wall of the left auricle), provided the conditions are suitable, as in the present instance. Possibly the physical condition of the lung overlying the heart's apex may occasionally contribute to hinder the propagation of a murmur outwards in this position. All these circumstances, coupled with the manner in which the murmur itself is actually produced, favour the convection of this murmur to the auricular, rather than its conduction to the mitral area.<sup>1</sup>

In another series of such murmurs the position of maximum intensity is strictly limited to the true pulmonary area, viz. the second interspace close to the left edge of the sternum, right over the pulmonary artery, which is here normally so placed that one-half of its lumen is covered by the sternum, while the other half lies in the interspace just to the left of that bone. This series again falls to be subdivided into two categories: one associated with a perfectly normal condition of the pulmonary artery, and the other with an abnormal state of that vessel. I shall

<sup>1</sup> In reference to these views Hayden, in his work on *Diseases of the Heart and Aorta* (Dublin, 1875), p. 1001, has pointed out that a presystolic murmur is only rarely to be heard in the second interspace, and when audible there may be readily recognised by its relation to the first sound and the apex beat. This is perfectly correct, but has no bearing upon the occurrence of a systolic murmur in the region referred to. When he also says that "a murmur of mitral reflux should be readily distinguishable by its special localisation and diffusion," he speaks correctly, with this proviso, that there are many cases of mitral reflux in which the "special localisation" is not the mitral but the auricular area; but with these cases it is evident that Hayden had not made himself familiar.

consider these two categories in detail, pointing out the diagnostic phenomena peculiar to each, and their signification.

In the whole course of my experience of cardiac disease, I have met with nothing more remarkable, nor at first sight more inexplicable, than the co-existence of a loud systolic murmur in the pulmonary region with a perfectly normal heart. Such cases simulate aneurysm of the sinus of valsalva so closely in the roughness of the murmur, the distinctness of the second sound, and the presence of abnormal pulsation, as to be somewhat puzzling, and apt to mislead. The only two points in which these cases differ from aneurysm in this situation are, first, in the entire absence of pain, which we know is not always present in cases of aneurysm; and, second, in the feebleness of the pulsation compared with that of the heart, a sign of unquestionable value in the diagnosis of aneurysm, but not one that can be wholly nor at any time solely relied upon as excluding the idea of aneurysm.

Dr. H. Quincke<sup>1</sup> of Berlin has devoted considerable attention to the subject of these murmurs, and as in many respects his observations coincide with my own, I shall first tell you his explanation of these inorganic pulmonary murmurs before narrating my own cases. First, however, I may tell you Quincke has pointed out, and has related one case in proof, that in certain rare cases of mitral stenosis in which the right ventricle is hypertrophied but not dilated, while the pulmonary artery is dilated, a systolic murmur may be heard in the pulmonary area, due to the formation of fluid veins at the pulmonary orifice as the blood passes through the healthy but comparatively narrow opening into the dilated artery beyond. This is obviously a physically possible cause for such a murmur, but it must certainly be a very rare one. In the case in point the left auricular appendix was thrombosed and lay deep within the chest; the

<sup>1</sup> Beiträge zur Entstehung der Herztöne und Herzgeräusche, *Berliner klinische Wochenschrift* (1870), No. 21, S. 249; vide also *Edinburgh Medical Journal* (January 1871), p. 667.



auricular origin of the murmur from convection was therefore entirely precluded. Quinke has also related six cases in which there was a systolic pulmonary murmur with perfectly sound hearts and arteries, the only abnormality being a retraction of the lung from the base of the heart which was common to all the cases of this series. He supposes that in these cases the murmur is produced by compression of the pulmonary artery by the heart during its systole, fluid veins being formed as the blood passes through the compressed and narrowed portion of the artery into the uncompressed, and therefore comparatively dilated portion of the vessel beyond—a condition which, if it is proved to exist, must be a very efficient cause of murmur. Quinke bases his views in regard to this matter upon certain facts which he observed. First, he found that in all these cases the pulmonary artery could be felt and seen pulsating in its normal position, and this visible pulsation was always associated with a systolic murmur, a murmur that ceased to be heard whenever the pulmonary artery was again covered by lung and ceased to be perceptible to touch or sight. Second, although there was not always any very obvious reason why the lung should not overlap the heart, yet in most of the cases there was sufficient cause for this imperfect expansion of the lung. In one case pulmonary expansion was prevented by pregnancy, and the murmur disappeared on the occurrence of abortion; in another case there was pneumonia; the only assignable reason in the other cases being debility from various causes. So long as the imperfect expansion of the lung continued the murmur was audible, but so soon as the lung expanded more perfectly the murmur ceased. On measuring the chest in both conditions, Quinke found that on cessation of the murmur the chest expansion was greater by a few centimetres than when the murmur was audible. He also noted that absolute cardiac dulness diminished *pari passu* with the return of the normal lung expansion and the disappearance of the pulmonary murmur. Moreover, in

many of those cases forced expansion of the chest is sufficient to cause complete cessation of the murmur, for just so long as the breath can be held. Quincke has also anticipated certain objections to his theory, due to the fact that a similar systolic murmur is not always to be heard even when pulsation of the pulmonary artery can be both seen and felt, by referring to the fact that murmurs are not always to be heard even when all the conditions needful for their production are present, as so often happens in cases of mitral stenosis. And he has also pointed out that in certain cases any compression of the pulmonary artery sufficient to cause a murmur may be prevented by the conformation of the chest, and particularly by any considerable prominence of the cartilages of the second and third ribs.

You know very well that compression of any artery sufficient to narrow its calibre materially is capable of originating fluid veins and a murmur at the part compressed, provided the blood is sent through it with sufficient force to make these fluid veins sonorous. If, then, we can connect retraction of the left lung from the base of the heart with a systolic pulmonary murmur, which disappears when the heart is more fully covered by deep inspiration, or by the natural removal of the cause restricting its expansion, and which may even remain inaudible notwithstanding the persistence of pulmonary retraction whenever compression of the pulmonary artery by the heart during its systole is by some cause or other precluded, then I think you will agree that in such a case Quincke's explanation seems a very probable one,<sup>1</sup> provided always there is no other apparent

<sup>1</sup> Quincke's idea is that in the normal condition part of the ventricular force during its systole is expended in forcing aside the overlapping edge of the left lung; with the lung already retracted, and the heart abnormally close to the chest wall, he supposes this portion of the systolic force to be employed in compressing the pulmonary artery. Hayden, *op. cit.* p. 1002, argues that if this were true we ought to have a murmur in every case of visible pulsation of the pulmonary artery. Quincke has anticipated this objection and sufficiently answered and disposed of it. Hayden has also very properly pointed out that in many cases of mitral stenosis we have marked

source of cardiac murmur to be detected. In the case now to be related you will find all these postulates present; and whatever difficulties may lie in the way of accepting Quinke's explanation, it seems still more difficult to account for the occurrence of such a murmur in any other way consistent with the physical laws bearing on the production of sound.

CASE XXIII. William M'Leod, aged thirty-two, formerly a soldier, now a van-driver, was admitted into Ward V. of the Edinburgh Royal Infirmary on 20th May 1870. He complained of an occasional sensation of beating in the left breast, accompanied by great breathlessness. These symptoms were liable to be brought on by exertion, and were always much increased by it. While a soldier in India some years previously, he had been laid up for a month with what appears to have been, from his own account, an attack of rheumatic fever; he also had syphilis there about fourteen years ago. His pupils were natural; his radial pulses unequal, the left radial pulse being a shade smaller than the right; the humeral pulses were, however, alike. On percussing the cardiac region transversely in the nipple line (along the upper border of the fourth rib), dulness was found to extend from the left edge of the sternum for a distance of one inch and a half to within half an inch of the nipple. On percussing in the parasternal line one inch from the left edge of the sternum from the clavicle downwards, dulness was found to commence at the upper margin of the second rib, and it extended down to the liver dulness (left lobe). The apex of the heart beat between the fifth and sixth ribs just below the nipple (two inches from the left edge of the sternum). Between the second and third ribs on the left side dulness extended for rather more than an inch from the left edge of the sternum, and within this space a pulsation

pulsation in this region not always accompanied by a murmur, and he supposes that this pulsation is due to "dilatation and distention" of the pulmonary artery. But the position of the pulsation is quite to the left of the pulmonary artery, and could only be produced by a large pulmonary aneurysm. In such cases we find after death a dilated auricle, and never a pulmonary aneurysm (*vide* p. 164).

was to be felt, distinctly less forcible than that of the heart. On auscultating in the mitral area a loud, rough murmur was heard just preceding and running up to the first sound, a presystolic murmur occupying the time of the auricular contraction. The first sound in this area was not pure, but there was here no systolic murmur; the heart's action was regular. Between the second and third ribs, just over the pulsation already noted, a loud and somewhat rough systolic murmur was heard closed by an accentuated second sound. This murmur was propagated on the left side down to the fourth rib, and also across the sternum to the space between the second and third ribs on the right side, where the aortic valve was heard to close with distinctly less force. The systolic murmur in the pulmonary area always became faint, and occasionally inaudible, when the patient assumed the erect posture; the presystolic murmur remained unchanged. The diagnosis of this case was mitral stenosis certainly, and probably also aneurysm of the ascending aorta, arising anteriorly just above the valve and passing to the left as aneurysms in this position generally do. The diagnosis of aneurysm was based on the presence of dulness and a pulsating tumour between the second and third ribs on the left side, and the fact that over this pulsation there was to be heard a loud, rough systolic murmur followed by an accentuation of the second much more marked than that usually heard in this position even in cases of mitral stenosis. The idea that this systolic murmur was of mitral origin propagated upwards by convection was rejected: first, because there was no murmur in the mitral area itself (at that time I was unaware that a mitral systolic murmur is often to be heard in the auricular area even though entirely absent from its normal position); and, second, because of the exceedingly loud and rough character of the murmur and its wide area of propagation. The "probably" was prefixed because we had seen so many similar cases that it was evident that either aneurysm in this position was more common than is usually supposed,



or there was some fallacy connected with the diagnostic signs, and I was inclined to hold the latter opinion. This patient was discharged, greatly improved, on 20th July. Previous to his discharge he was seen and examined by the late Professor Sanders, who confirmed the diagnosis in every respect and especially as to the "probability" of its being a case of aneurysm. M'Leod was readmitted on 16th October complaining of irregular attacks of ague, from which he had formerly suffered in India; for this he had been taking full doses of arsenic for a week without benefit. He was ordered five grains of quinine every two hours, and under this treatment he rapidly improved. On the evening of 27th October he had a severe rigor, followed by fever and a restless night. On the 28th he was still feverish, with quick respirations, rusty gelatinous sputa, and fine crepitation over the right lung posteriorly; there was no dulness. On the 29th he was much feebler, the respirations 60, otherwise as yesterday, and in spite of free stimulation he died at 3 P.M.

*Autopsy.*—1st November. On opening the thorax the left lung was seen to be retracted upwards and backwards, receding from the mesial line opposite the second and third ribs; it dipped down opposite the fourth and fifth ribs, leaving uncovered the whole of the right ventricle, the apex and lower half of the left ventricle, and to a slight extent the pulmonary artery also. The amount of serum in the thoracic cavity was normal. Both lungs were deeply congested, and floated heavily—especially the right one—but neither were properly speaking hepatised. The heart weighed  $19\frac{1}{2}$  oz., and was hypertrophied and slightly fatty. The pulmonary and tricuspid valves were healthy. The cusps of the aortic segments were thickened, and the valve itself slightly incompetent; the aorta was healthy. The mitral valve was much thickened, especially its aortic segment; the left auriculo-ventricular opening was so contracted as only to admit the tip of the little finger; the *chordæ tendineæ* of the mitral valve were thickened. There was some cretaceous deposit at

the junction of the auricle and ventricle. The liver weighed 4 lbs., and presented an indistinct nutmeg appearance; its capsule was healthy. The kidneys were pale and fatty, the cortical part increased one-third in volume; the capsules free, the surface beneath smooth. The spleen weighed 2 lbs.  $1\frac{1}{2}$  oz.; it was softened, and on section presented a few hæmorrhagic spots from the size of a shilling downwards.

The autopsy, while confirming the diagnosis in every other particular, gave no support whatever to the idea of an aneurysm, as not a trace of such a lesion was to be seen. For this, however, I was fully prepared by the occurrence of the following still more remarkable case:—

CASE XXIV. James Morrison, a bricklayer, aged thirty-one, admitted to Ward V. on 25th April 1870, complaining of soreness in the chest, headache, dyspnœa, and some swelling of the face and body generally. I shall omit the general history of the case, which was an ordinary one of Bright's disease, merely premising that the patient was by no means anæmic, though of a somewhat sallow complexion, and that the dropsy present was merely an inconsiderable amount of general anasarca, depending upon an early stage of cirrhosis of the kidneys, with slight traces of amyloid degeneration, as ascertained by inspection after death. Morrison was a powerfully built man, and all his other functions and organs were perfectly healthy with the exception about to be mentioned. His pupils were both equal, and both pulses equal at the wrist. The apex of his heart beat between the fifth and sixth ribs, just outside the nipple line, and was somewhat diffuse. In the parasternal line one inch to the left of the sternum dulness commenced at the lower edge of the second rib and extended down to the liver dulness. In the nipple line (along the upper border of the fourth rib) dulness extended transversely from the left edge of the sternum to the left nipple, a distance of three inches. Between the second and third ribs on the left side there was an evident pulsation, which was most distinct on deep expiration. This pulsation

extended for three-quarters of an inch from the left edge of the sternum, and ended quite abruptly; to the left of this the dulness continued to be marked for quite two inches, but it scarcely encroached at all upon the sternum. On auscultation in the mitral area the first sound was heard to be rather feeble, but distinct and without murmur; the second sound was somewhat accentuated. Between the second and third ribs at the right edge of the sternum the first sound was still heard somewhat faint, but free of murmur, while the second was more markedly accentuated, and this accentuation of the second increased as the stethoscope was moved across the sternum till it attained its maximum over the pulsation already described. At this point, between the second and third ribs on the left side close to the sternum, the accentuated second was preceded by a particularly loud and rough systolic murmur, very well marked and distinct, occupying the position but presenting a striking contrast to the soft, blowing, so-called hæmic murmur. This murmur could be faintly heard over the innominate and right carotid arteries; but not a trace of it was to be heard over any of the other vessels, though the accentuated second sound was more or less distinctly audible in both subclavian and in both carotid arteries. It is scarcely possible to imagine any objective signs which could more closely resemble those of an aneurysm arising from one of the sinuses of valsalva and passing to the left: with this single proviso, that the pulsation was less forcible than that of the apex beat; and this cannot after all be accepted as sufficient negative evidence. Subjective symptoms either of aneurysm or of any cardiac disease were entirely absent. The absence of subjective symptoms is certainly not sufficient to entirely discredit the existence of an aneurysm, but it would have thrown a halo of suspicion around any case with signs less well marked than this one appeared to possess.

While still under treatment, Morrison was unfortunately seized with an acute attack of erysipelas of the head, to which he succumbed upon 29th June. At the autopsy

on 1st July the left lung was found to be somewhat retracted, leaving the base of the heart and the pulmonary artery rather more uncovered than usual. The aorta was found to be very slightly dilated, but the heart itself was normal in every respect, except that the left ventricle was slightly enlarged (dilated and hypertrophied). The pulmonary artery was perfectly normal and its valves healthy. The interior both of the aorta and of the pulmonary artery, as well as that of their chief branches, was perfectly normal, smooth, and wholly free from every trace of atheroma.

CASE XXV. J. W.,<sup>1</sup> aged twenty-eight, a fish salesman, admitted 18th June 1874, to Bed 7, Ward V. of the Edinburgh Royal Infirmary, complaining of lassitude, weakness, and pain in the abdomen. About six months previously he had pain in the chest, severe cough, night sweats, and loss of appetite, brought on by exposure to cold and wet. These symptoms in a great measure disappeared under treatment, but since then he has always had some cough and has never regained his strength. About a month previous to admission he first noticed a swelling in his right side, which was painless itself but accompanied by pain in the stomach after every meal, and occasionally at other times also. The patient has always been healthy, except that in 1869 he passed through an attack of cholera, and about a year subsequently he had a fever, nature unknown. His family history is good; his father and mother, three brothers, and two sisters are all alive and in good health. The patient states that he has never been abroad, has never suffered from ague, rheumatism, or syphilis; he has never had any suppurating wound or sore, beyond a trifling scratch; but he has been in the habit of drinking beer and other malt liquors in large quantities. On admission his muscles were fairly developed, but his face was thin and his expression anxious. His temperature was 99°, and his respirations 16 per minute. He had night sweats, and occasional cough, especially at night. The hepatic region

<sup>1</sup> Reported by W. R. Gibson, clinical clerk.



was tumid but soft, and there was considerable pain on pressure just above the umbilicus.

On 3rd July the patient had emaciated somewhat, his muscles were soft and flabby, night sweats continued. Since admission his temperature has varied from  $97^{\circ}$  to  $104^{\circ}$  in a most irregular and capricious manner. Only once it has been  $104^{\circ}$  (on the evening of 26th June), and it was only  $98^{\circ}$  on the preceding and on the following morning; the average temperature has been little over  $100^{\circ}$ ; his respirations have also varied, the average being about 20 per minute. He coughed severely at times, chiefly during the night. The thorax was well formed, and expanded equally on both sides. Percussion was normal over both lungs. On auscultation rough vesicular breathing was heard over both lungs, both in front and behind, mingled with an occasional mucous rattle or a rhonchus. The pulse was of fair strength, 90 per minute; the peripheral arteries and veins were normal. No cardiac pain or palpitation was complained of; but on inspection the heart's pulsation was more apparent than usual, and between the second and third ribs on the left side, close to the sternum, an unusual but distinct pulsation was observed, and this seemed to propagate itself upwards to the root of the neck as a slight tremor running superficially over the parieties. At one inch from the left edge of the sternum dulness commenced at the lower border of the second rib and extended downwards to the liver dulness. In the intermammary line dulness commenced at the right edge of the sternum and extended across for a distance of four inches. In the second interspace dulness extended to the left of the sternum for a distance of two inches and a half. In the mitral, tricuspid, and aortic areas the normal heart sounds were distinctly audible. In the pulmonary area the first sound was entirely replaced by a loud, rough murmur, followed by a more than usually distinct but not accentuated second sound.<sup>1</sup> This systolic

<sup>1</sup> There was no marked congestion of the lungs, such as we have in mitral stenosis, therefore the element of tension was wanting; *vide* p. 31.

pulmonary murmur was found to disappear completely when the patient took a deep respiration and held his breath, reappearing at once on expiration; it was not propagated in any direction. The tongue had a white coating, with a triangular red patch in the centre; the apex of this coincided with that of the organ. The patient's appetite was impaired, and he had a feeling of oppression in the *scrobiculus cordis* after meals, with much thirst. The bowels were irregular, most commonly loose. The abdomen appeared distended, and had a peculiar tense, elastic feeling when palpated. There was slight tenderness on pressure all over it, and this culminated in positive pain at a point just above the umbilicus in the mesial line. Round the umbilicus the patient measured thirty-one inches and a half; three inches higher he measured thirty-three inches and a half: this increase was very evidently due to bulging of the liver, which was enlarged, hepatic dulness in the mammary line measuring seven inches. Splenic dulness was somewhat increased. Micturition was more frequent than usual, and occasionally accompanied by pain along the course of the urethra. The urine was of a dark straw colour, acid, specific gravity 1015, and averaged from 50 to 60 oz. daily. On standing there was a considerable deposit of mucus, and it contained a large quantity of albumin, but no sugar or bile. On microscopical examination the deposit was found to consist chiefly of amorphous matter, with epithelium and a few fragments of hyaline and slightly granular casts.

The subsequent history of this case was that of gradual emaciation and sinking, with an irregular temperature averaging about  $100^{\circ}$ ; falling sometimes to  $97^{\circ}$ , and again rising to  $102^{\circ}$ . There was persistence and a gradual increase of the diarrhoea; the urine continued highly albuminous, never under 35, and sometimes as high as 90 oz. in the day. There was also a gradual but trifling accumulation of fluid in the abdomen and in the cellular tissue of the feet and legs; and latterly a very slight degree of jaundice was developed.

On 13th July it was noted that the painful spot above the umbilicus had become exceedingly tender to touch; nothing special could be detected there, but low down in the right iliac region a small limited hard patch was to be felt at the lower border of the liver.

On 21st July the systolic pulmonary murmur had entirely disappeared, and it was not again heard.

The patient gradually sank and died on 30th July.

*Autopsy.*—Body much emaciated; skin and conjunctivæ somewhat jaundiced. There were pleuritic adhesions extending down the whole of the left side of the thorax at the junction of the ribs with the cartilages. The fibrous sac of the pericardium was more fully exposed than usual, apparently due to the lungs having collapsed very markedly when the chest was opened. The heart was small, weighing 7 oz.; all its chambers contained decolorised blood-clot. On the anterior surface of the right ventricle there was a milk spot, which measured one inch and a half in length, and three-quarters of an inch in breadth. The thickening of the pericardium at this spot was unusually marked. The chambers of the heart were all of the natural dimensions. The tricuspid and mitral valves were quite healthy; the tricuspid orifice admitted four fingers, the mitral three. The aortic and pulmonary valves were quite competent and otherwise natural. A tough, decolorised blood-clot occupied the pulmonary artery, and extended along its primary ramifications into both lungs. The aortic wall was free from atheroma and otherwise natural. On microscopical examination the muscular fibres of the heart were found to be perfectly healthy. The left lung weighed 14 oz.; the right, 14½ oz.; both were somewhat congested in their posterior three-fourths, and there was a little emphysema at the anterior edges of both. The peritoneal cavity contained rather more than two quarts of turbid serum, in which floated numerous flakes of soft recent lymph. The liver weighed 6 lbs. 1½ oz.; its surface was smooth, but its

capsule presented ramifying lines of white opacity, and in the upper surface of the right lobe there was an opaque thickening with puckered stellate edges like a cicatrix. The thickening did not, however, extend beyond the capsular surface. On section, the organ presented a slightly granular aspect, and felt exceedingly tough and fibrous. It also presented to the naked eye some of the characteristic appearances of waxy degeneration, and it gave the usual staining on the application of the iodine test, but not very markedly. On microscopical examination a very marked increase of connective tissue was observed, both within the lobules and at their peripheries. The pancreas was of natural size and healthy texture; it weighed, with a few lymphatic glands attached to it,  $4\frac{1}{2}$  oz. Close above the margin of its head and upper part of body there were a great many enlarged lymphatic glands, varying in size from a pea to almost that of a pigeon's egg. From the pancreas a string of these glands extended up to the liver, between the folds of the gastro-hepatic omentum. The mesenteric and prevertebral lymphatic glands around the coeliac axis, and for two inches and a half below, were enlarged, and had undergone a degeneration similar to those above the pancreas. On microscopical examination of these glands, the lymph-cells were found to have run together into waxy translucent clumps, which took on the waxy reaction very markedly, as did also the vessels connected with them. There was no obstruction of the cystic or hepatic ducts. The spleen was much enlarged; it weighed  $13\frac{1}{2}$  oz. Its capsule was firm and fibrous, and the Malpighian corpuscles were unnaturally prominent. These corpuscles were very markedly stained when the iodine test was employed. The kidneys were both enlarged; the right weighed 8 oz., the left 7 oz. The cortical substance was much increased, and the organs were very much congested. The Malpighian bodies were readily stained by iodine. The mucous membrane of the intestines presented very markedly the waxy reaction.



You will observe that these cases all agree in this, that there was in each of them marked retraction of the left lung, with uncovering of the heart and pulmonary artery. The exposure of the pulmonary artery permitted its pulsation to be readily seen and felt. This pulsation could not possibly be mistaken for that of a dilated auricle, inasmuch as it occupied quite a different position close to the sternum, and was both seen and felt to terminate abruptly just as the auricular area was reached. Moreover, the finger resting on the pulsation could in every case distinctly feel the click of the closure of the pulmonary valves, so that the arterial origin of the pulsation could scarcely have been doubted even had the left auricle been simultaneously dilated, which was not the case in any of the examples given except the first, and even in it the dilatation was but trifling. The uncovering of the pulmonary artery brings the valve closer to the surface; hence its closure is not only readily perceived by the finger, but it is also more distinctly heard, and thus accentuation is simulated even when it is not actually present. In the second case narrated (Case XXIV. p. 226) there was slight dilatation of the aorta, too limited in extent to be readily made out by percussion, but undoubtedly sufficient to cause accentuation of the aortic second, as well as a systolic murmur propagated into the arteries of the neck. These facts, coupled with the greatly increased intensity of the second sound over the pulsation in the second left interspace, as well as the peculiar localised roughness of the systolic murmur which preceded it, complicated the case and made it to be long regarded as an aneurysm of one of the sinuses of Valsalva. Indeed the peculiar loudness, roughness, and localised character—not propagated in any direction—of this murmur make it in every respect one of the most remarkable in the history of cardiac disease. In the cases narrated there is no reason to doubt that the spanæmia present in all of them assisted in the causation of the murmur. It will be found noted in regard to the case

first described (Case XXIII. p. 223) that the murmur disappeared whenever the patient assumed the erect position, probably because in this posture the compression of the pulmonary artery by the heart was prevented by the weight of the heart dragging it down. In the third case (Case XXV. p. 228) the systolic murmur in the pulmonary artery disappeared entirely whenever the patient took a deep inspiration, and did not reappear until he expired. Moreover, nine days before the patient's death, when the heart must have been somewhat atrophied and was certainly enfeebled, and therefore not only less able to compress the pulmonary artery, but also less able to drive the blood through it with a force sufficient to produce a murmur, then this murmur ceased for ever. It seems impossible to account for the retraction of the lung in the two first recorded cases. In the third case there were two efficient causes contributing to prevent complete covering of the heart during ordinary inspiration: first, there was the enlargement of the liver and the pain in the abdomen, and, second, there was the string of interpleural adhesions along the junction of the ribs with the cartilages on the left side; these, though not tight enough to prevent complete covering of the heart on forced inspiration, no doubt contributed their quota in preventing complete covering in the ordinary restrained respiration of this patient. In a fourth case, of which unfortunately I cannot give full details, the cause of the retraction of the lung was only too apparent. This patient was for some time under my care in Ward XIII., presenting all the ordinary phenomena of pulmonary phthisis, affecting mainly the upper lobe of the left lung. At that time she had no cardiac murmur whatever. She improved considerably, and was discharged. About a year afterwards she was admitted into Ward XV. under the care of my colleague, Dr. Haldane, and he directed my attention to her as a remarkable case of pulmonary murmur. In this case there had been several small cavities in the upper lobe of the left lung; as these

cicatrised the lung contracted, and *pari passu* with its retraction from the base of the heart there was developed in the pulmonary area a loud, rough, localised systolic murmur. These cases do not prove the correctness of Quincke's theory, but they lend a strong support to it, and it is difficult to conceive another theory equally simple and equally probable, which will harmonise so well with all the facts associated with this localised murmur as we find it in nature.

The systolic murmurs associated with an abnormal pulmonary artery need not detain us long. These cases are mostly congenital, and always rare; they are more objects of medical curiosity than of medical treatment. They are mostly congenital, and as a rule each individual case presents, besides the ever-present murmur or murmurs in the pulmonary artery, other murmurs and signs of co-existing congenital malformations, which vary in each case. The following case is the only one of the kind that occurred to me during my connection with the Infirmary:—

CASE XXVI.<sup>1</sup> Anne C., aged nineteen, a dressmaker, born at Aberdeen, and residing at Leith, was admitted to Bed 10, Ward XIII., on 1st December 1873, complaining of great pain over the præcordia, extending to the left back and shooting down the left arm, and of a bad sore throat. The patient is usually deeply cyanosed, but her complexion varies; the digits on all four extremities are clubbed in a very marked degree and the nails aduncated.

Her mother stated that last February (1873), after a fright, her daughter was seized with a sudden pain in the left side, which finally settled over the præcordia. At that time she suffered much from palpitation and a feeling of general oppression in the chest. This state of matters lasted till May. After a short period of comparative health she was seized in June with a shivering fit, and shortly after coughed up two mouthfuls of dark blood, and several more of a lighter colour. Since then she has been always ailing. Her mother

<sup>1</sup> Case reported by Mr. Kelly, clinical clerk.

also stated that ever since birth she had been of a blue colour, most marked after exertion and in cold weather. Shortly after her birth her father contracted syphilis, which he communicated to her mother, and through her mother to herself and to all the children born subsequently. So that all the children born previously are healthy, while she and her mother suffer from acquired syphilis, and all her brothers and sisters born subsequently suffer from congenital syphilis.

Anne C. is fairly developed, rather above the average height; she weighs 8 stone 3 lbs. Her expression is dull; her complexion rosy, sometimes livid. Her lips are generally livid, and the tips of her clubbed fingers are always so, and the same may be said of her ankles and toes. Her skin generally is not discoloured; it is sometimes warm, moist, and clammy, at others cold and dry. Her temperature is  $98.4^{\circ}$ . Her pulse varies from 68 to 75 per minute; it is feeble and equal at both wrists. The jugular veins are small, but pulsation is distinctly visible in them. She has dyspnœa on the slightest exertion, no palpitation, but severe pain over the præcordia. On inspection the chest appears normal; no pulsation is anywhere visible. On palpation no impulse is perceptible, even when she leans forward; but a considerable thrill is felt over the base of the heart. On percussing in the parasternal line one inch to the left of the sternum dulness begins at the upper edge of the third rib, and extends down to the liver dulness. At the level of the fourth rib transverse dulness begins three-quarters of an inch to the right of the sternum, and extends to the left for a distance of four inches and three-quarters. The aortic dulness extends upwards to within one inch of the top of the sternum. On auscultation a loud systolic murmur is heard over the whole of the cardiac area, and it is propagated with more distinctness towards the left than towards the right axilla. Careful auscultation at the back of the chest enables us to recognise a faint systolic murmur, evidently due to propagation through the bones. In the mitral area (two inches and a half to the



left of the sternum in the fifth interspace) this systolic murmur is shrill and distinct, followed by a faint second sound. In the tricuspid area (the sternal end of the fourth, fifth, and sixth ribs on the left side) the systolic murmur is louder, more prolonged, and followed by a more distinct second sound. In the aortic area (sternal end of the second rib on the right side) the systolic murmur is shrill, distinct, and followed by a clear second sound, often markedly accentuated; both murmur and sound are propagated into the carotids. In the pulmonary area (the sternal end of the third rib on the left side) the systolic murmur is louder and rougher than at any other part of the cardiac area, and it is followed by a second sound which is always distinct, and at times is quite accentuated. When the pulmonary second is best marked the aortic accentuation is least distinct. The patient's respirations are 20 per minute, and she has dyspnoea on the slightest exertion; there is also pain in the chest, but no cough or expectoration; the pulmonary physical signs are normal. Her voice is hoarse. Apart from sleeplessness, occasional darting pain over the left temple, and the præcordial pain already referred to, her nervous system is quite normal. She has considerable difficulty of swallowing from syphilitic congestion of the uvula, the tonsils being also swollen and congested; there is also slight atonic dyspepsia, otherwise the digestive system is normal. The urine is of a pale sherry colour; it is acid, specific gravity 1020, and contains a small amount of albumin; it is otherwise normal; there is no deposit. Menstruation is regular.

This concisely narrated history contains all the elements needful for an accurate diagnosis, so far as that is possible. The history and the symptoms of acquired syphilis, sufficiently distressing in themselves, are yet purely episodic in relation to the more serious affection under which this patient suffers, an affection which, from its congenital character, has necessarily embittered all her life, and must ultimately shorten her days. The cyanotic condition of her surface, and the

remarkable clubbing of all her digital extremities, point with unerring accuracy to the existence of some congenital central lesion of her circulation. In regard to the production of cyanosis, three views are prevalent—first, there is Morgagni's view that it depends upon general congestion from some obstruction to the onward flow of the blood; second, there is Hunter's idea that it is caused by an intermixture of the arterial and venous blood currents; and there is a third view adopted by some who think that there must be both intermixture and congestion before marked cyanosis can be developed. This latter view seems most consistent with the facts as observed in nature. Clinical experience teaches us that even when the obstruction to the onward flow of the blood is very great, the cyanosis is never so great as in those cases where to congestion from any cause there is superadded, from congenital defect, a possible intermixture of the two kinds of blood. It is almost impossible to imagine greater obstruction to the circulation than what must have existed in the patient to whom the mitral valve—now before us—belonged; in it, as can be seen, the opening, somewhat enlarged by long maceration in spirit, only measures five millimetres by eight; yet she had no marked cyanosis; her lips alone were somewhat dusky. On the other hand there are many cases on record in which intermixture of the two kinds of blood was at least possible, and yet in them also there has been no cyanosis.<sup>1</sup> Of this we may be, however, certain, that whenever we have a case of marked cyanosis there is always present one or more of those congenital lesions which make possible this admixture of arterial and venous blood. In the present case this opinion is confirmed by the clubbing of the digital extremities—a condition that must have a cardiac origin if it extends, as in this case, to all four extremities in any marked degree. These conditions, therefore, enable us

<sup>1</sup> Many such cases are on record. I may refer specially to Rokitsky's *Handbuch der pathologischen Anatomie*, Bd. ii. (1844), S. 513, etc.; and also his work *Die Defecte der Scheidewände des Herzens* (Wien, 1875).

to conclude with certainty that here we have to do with a congenital malformation of the heart capable of permitting intermixture of the arterial and venous blood. Now, apart from congenital deficiencies of the septa alone—which to any considerable extent are extremely rare, cause little if any cyanosis, and either give rise to no murmurs or to murmurs perfectly different in character and mode of propagation from those in this case—we learn from the researches of those pathologists who have made a study of cardiac malformations that the probability of the original lesion being in the pulmonary artery increases as the age of the sufferer exceeds fifteen. The age of our patient is nineteen; there is therefore a strong probability from that fact alone that her primary lesion is in the pulmonary artery. And this is confirmed by the presence of the rough systolic murmur audible in the pulmonary area, and propagated with most distinctness towards the left axilla. The distinct and accurate closure of the pulmonary valve (absence of diastolic murmur) shows us that the murmur probably depends upon a constriction lying above this valve, as, if its segments were so malformed as to obstruct the onward flow of the blood, they would almost of necessity be incompetent also. But the necessary result of constriction of the pulmonary artery to any extent, or for any lengthened period—long-continued action being equivalent to greater constriction—would inevitably result in a gradually increasing congestion and dilatation of the right ventricle. Of this in the present case we have ample proof in the loud systolic murmur audible over the right apex (in the tricuspid area), and in the distinct jugular pulsation, which shows that the tricuspid regurgitation has been so considerable and so long continued that it has destroyed the action of the venous valves at the root of the neck, and has thus converted what must at first have been a mere undulatory movement in the venous current into a distinct pulsatory wave. The presence of so great a dilatation of the right ventricle sufficiently explains the absence of an apex beat, because the dilated

right ventricle lying in front of the left one pushes the apex from the chest wall into the thoracic cavity (*vide* p. 199). The absence of any pulsation in the *scrobiculus cordis*, and at the lower part of the sternum, shows that the heart is feeble, and that dilatation is the prevalent lesion of the right ventricle.

Without entering into any exhaustive disquisition on the subject, it may suffice to say that constriction of the pulmonary artery may take place at various periods of intra-uterine life, and the mechanical results on the development of the heart must vary accordingly, and with them *pari passu* also the signs and symptoms. When such a constriction occurs just previous to the closure of the ventricular septum, which is thus prevented, we have as a natural result a complete double circulation with free intercommunication between the two ventricles, one of which is connected with an obstructed and the other with a patent arterial conduit. As both ventricles contract with nearly equal force upon nearly equal contents, the result must be that the ventricle with an obstructed outlet must force part of its contents through the abnormal channel into the other ventricle whose outlet is unimpeded. The consequence of this is forcible dilatation of the unimpeded conduit—in this case the aorta—and consequently a systolic murmur of distention accompanying the blood-wave, followed by a loud accentuated second sound, the natural result of the forcible closure of the aortic valve by an unusually heavy column of blood—precisely the conditions and the signs usually present in this case. When from any cause, however, the pulmonary circulation is more than usually obstructed, as from catarrhal congestion of the lungs, then, as in the normal condition, the blood-recoil upon the pulmonary valve is greatly increased, and we have a temporary transference of the accentuation from the aortic to the pulmonary valve—a phenomenon which repeatedly occurred in this case, under the conditions described, while she remained under observation.



The systolic murmur in the mitral area is in all probability due to propagation from the aortic and pulmonary areas, possibly reinforced by the murmur due to the passage of the blood through the abnormal opening in the ventricular septum, which I have shown is most probably present. It is not likely to be due to mitral regurgitation, as there is no distinct propagation round the left side to the back, and with a free communication between the two ventricles there is an entire absence of the conditions needful to produce overdistention of the left ventricle and regurgitation through the mitral opening. A mitral stenosis, and regurgitation from this cause, would be a most unusual complication in such a case as the present, and is further rendered highly improbable by the entire absence of any dilatation of the left auricle or of its appendix.

In this most interesting case, therefore, there seems conclusive evidence of the presence of contraction of the pulmonary artery above the valve, dilatation of the right ventricle with incompetence of the venous valves at the root of the neck, deficiency of the upper part of the interventricular septum, and dilatation of the ascending aorta. There is also a more or less imperfect occlusion of the *foramen ovale*. We know of no signs by which this may be recognised, but from what we know of the sequential development of the heart this abnormality may be accepted as a necessary complement of the other conditions present.

A mere cursory inspection of this case would lead almost any one to conclude that she laboured under serious congenital malformation of the heart, and it is interesting to note how the physical signs present, coupled with our knowledge of the mode in which these physical signs are produced, have enabled us to follow up the clues thus suggested till we have been able, with the highest probability, to identify the primary lesion as a stenosis of the pulmonary artery.

I have only to add that this patient was able to leave the Infirmary improved in every respect. For some years she

came occasionally under observation, her signs and symptoms remaining unchanged.

An opening between the two ventricles in cases of pulmonary stenosis is rather favourable to longevity than otherwise, and though twenty years is but seldom attained by such patients, some have been known to live to thirty and forty, and one case of undoubted stenosis of the pulmonary artery has been known to live to sixty-five.<sup>1</sup> The signs in every case vary with the nature and extent of the malformation, but this can always be ascertained with greater or less probability by cross-examining the various signs and symptoms somewhat after the fashion just described.

In some cases of congenital malformation of the pulmonary artery, and more rarely as the result of disease after birth, the pulmonary valve is incompetent, and instead of a second sound we have a diastolic murmur. Dr. Warburton Begbie has published a case he met with in this Infirmary.<sup>2</sup> In it the pulmonary valve consisted of four segments instead of three. This incompetence of the pulmonary valve was diagnosed during life, and the diagnosis confirmed by examination after death, which was the result of accident. Incompetence of the pulmonary valves must be rarely seen after childhood, but it does occur as the result of general or local disease. It is not difficult to detect, but we must be careful not to mistake a diastolic mitral murmur<sup>3</sup> for a diastolic pulmonary, or the converse. The diastolic murmur occasionally found in connection with mitral stenosis often has its position of maximum intensity at the sternal end of the fourth rib, and is naturally liable to be mistaken for either an aortic or a pulmonary diastolic murmur. It is sometimes very difficult to be certain that there is no aortic

<sup>1</sup> *Vide* Kussmaul, "Angeborene Enge und Verschluss der Lungenarterienbahn," *Zeitschrift für rationale Medizin* (Leipzig, 1866); and Peacock, *Malformations of the Human Heart* (London, 1866), 2nd edition, etc.

<sup>2</sup> Beale's *Archives of Medicine*, No. 5; and *Warburton Begbie's Works*, by Sir Dyce Duckworth, New Sydenham Society (1882), p. 109.

<sup>3</sup> *Vide* Lecture V. p. 135.

regurgitation, and occasionally we may even have to wait further development before we are able to give a definite opinion. In all cases of pulmonary diastolic murmur hitherto recorded there has always been, I believe, a loud systolic murmur preceding it, so that there is less danger of any mistake being made in this direction.

Most of the cases of congenital affections of the pulmonary artery are complicated with some abnormality of the *ductus arteriosus*, which may either be imperfectly closed or aneurysmally dilated.<sup>1</sup> As a mere complication this abnormality is rather an advantage than otherwise, in some, indeed, contributing largely to the carrying on of the circulation. But as an independent affection it is of much more importance, and chiefly because the loud murmur which reveals its presence causes an unimportant imperfection to be invested with possibilities for the patient which loom all the more seriously on his future that they are not a little mysterious to the observer himself. I have only seen two cases in hospital in which aneurysm of the *ductus arteriosus* was the predominant lesion. Both were discharged *in statu quo*. One of them turned up subsequently in Glasgow, and her case has been published by Dr. Wood Smith.<sup>2</sup> The only case of the kind in which I know death to have occurred was one, also complicated with other malformations, which I saw about twelve years ago with Dr. James Foulis, under whose care she was.<sup>3</sup> But cases of uncomplicated open *ductus arteriosus*, frequently more or less aneurysmally dilated, are common enough and important enough to deserve an endeavour to differentiate them from other affections with which they are often associated and frequently confounded.

About twenty years ago I was asked by the late Dr.

<sup>1</sup> Vide Rokitansky, *Ueber einige der wichtigsten Krankheiten der Arterien*, (Wien, 1852).

<sup>2</sup> Vide *Glasgow Medical Journal* (August 1879), p. 103.

<sup>3</sup> Vide "On a case of patent *ductus arteriosus*, with aneurysm of the pulmonary artery," by James Foulis, M.D., *Edinburgh Medical Journal* (July 1884), p. 17.

Warburton Begbie to visit and report upon the case of a young lady who had been seen about ten years previously by his father, Dr. James Begbie, on account of some peculiar heart symptoms. I reported that I could find nothing wrong with her heart, and that I believed she suffered from an open *ductus arteriosus*, which was probably aneurysmally dilated. I was then told that both Begbies—father and son—had independently arrived at a similar conclusion. The patient was also seen and independently examined by the late Professor Sanders, who entirely homologated our views.

This patient had considerable venous congestion of the surface generally, her face was dusky, her fingers and toes quite purple, and her extremities always cold. She complained of breathlessness, increased on exertion, and of cough without expectoration. The heart was normal in all its dimensions, but there was distinct pulsation between the second and third ribs to the left of the sternum. Over the whole of the cardiac area a loud systolic murmur was heard, and this was propagated into the arteries and very distinctly audible down the back along the course of the descending aorta. The position of maximum intensity of the systolic murmur was over the pulsation between the second and third ribs to the left of the sternum, where it was accompanied by a loud purring, revealed to a palpating hand as a purring thrill—*frémissement cataire*—and radiating round the pulsation as a centre, but more strongly upwards in the course of the vessels. This loud purring systolic murmur was closed at the moment of diastole by a loud metallic clang, most distinctly heard close to the pulmonary area.

The main elements in the diagnosis of this case were, first of all, the length of time the signs and symptoms had remained unchanged; second, the early period of life at which they had made their appearance; third, the position and character of the murmur present; and, lastly, the manner in which the symptoms varied under the influence of circumstances producing alterations in the relation between the



systemic and pulmonic circulations. I have seen this patient repeatedly; she has continued to improve, and the last time I saw her, some four or five years ago, there was neither duskiness of complexion nor any murmur left; to appearance and on careful examination nothing was to be detected in her different from any other young woman of her years. Evidently the *ductus arteriosus* had completely closed, as it is occasionally known to do even late in life, probably from occlusion of its lumen by a clot which becomes organised. Aneurysm of the *ductus arteriosus* is not a great rarity; I see a case every now and then, sometimes still in early youth, more often young adults who have been baulked in some object in life on account of the detection of this remarkable murmur. Where the malformation is limited to the open duct, no complaint is ever made and no suffering experienced: there is seldom even a trace of cyanosis; but there remains the limited pulsation in the second interspace, which, when palpated, communicates a vibratile thrill to the hand, a thrill which radiates upwards along the vessels, and upper part of the left chest. On auscultation over this pulsation there is always a systolic murmur, sometimes followed by a diastolic one; throughout both systole and diastole there is a continuous purring—*frémissement cataire*—and whether there is a double murmur or not the commencement of the diastole is marked by a loud metallic clang. These murmurs are audible always up the left side of chest and in the left carotid, and occasionally over the whole cardiac area, as well as in the right carotid artery, but naturally they vary in intensity and propagation in each individual case, yet always presenting the characteristic purring. These cases improve as life goes on, and, as we have seen, they sometimes get well. The only case in which death is known to have occurred is the one already referred to, in which other malformations hastened the end. In it, however, the diagnosis of an open *ductus arteriosus* was fully confirmed by the examination of the organs after death.

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The remarkable series of cases narrated in this lecture fully vindicates the right of the pulmonary area to be called the region of romance, yet all the murmurs audible in this region have each their own distinctive peculiarities, which, rightly interpreted, enable us to unravel their mode of origin and to discover their ultimate cause with greater or less probability according to the understanding and the skill brought to bear upon each individual case.

I have just one other remark to make in conclusion, and that is, that systolic pulmonary as well as aortic murmurs are occasionally produced by pressure from without from tumours, or more rarely from disorganised lung or intra-pericardial fluid. In all these cases we have the absence of all the signs and symptoms indicative of any of the other modes by which a murmur in this situation may be produced. Occasionally we are able by percussion to detect the presence of the morbid growth or altered structure. Now and then, however, the diagnosis is sufficiently obscure, must be made mainly *per viam exclusionis*, and is after all largely a matter of probability.

## LECTURE IX

### ON THE VARIATION AND VANISHING OF CARDIAC MURMURS

It not infrequently happens that a patient presents himself with a note from his medical adviser stating that So-and-So labours under cardiac valvular disease, and yet on careful examination we are unable to detect any murmur, and sometimes not even any other sign that would lead us to suspect the existence of a cardiac lesion. What are we to say to this? Are we wrong, or has the medical attendant been mistaken? Our experience in the wards supplies a sufficient answer to this important question; for I may safely say that scarce a day passes in which we have not an opportunity of observing the remarkable manner in which even murmurs dependent upon recognised organic lesions change and vary in character and intensity, and not infrequently disappear, the lesion still remaining. This is an important fact, and one too apt to be overlooked in the consideration of cardiac disease. It has led to many an unlucky *contretemps*, and, as I have already mentioned, to the awkward fact of a man dying of organic cardiac disease yet possessed of quite a bundle of certificates from competent medical authorities certifying that he had no cardiac disease whatever.<sup>1</sup>

But while ignoring the fact that murmurs undoubtedly depending upon incurable cardiac lesions may vary in character and even vanish has led to such untoward results, ignoring the equally important fact that many murmurs are

due to perfectly curable lesions has in my own experience led to results even more lamentable. Many years ago a patient of my own, dyspeptic and hypochondriacal, who had a slight systolic murmur of mitral origin due to simple dilatation of but trifling character, consulted a physician who led her to understand that she laboured under an incurable affection of her heart. Foolishly connecting this diagnosis with sudden death as the inevitable result of any exertion, she laid herself upon her sofa, and the greatest persuasion could never again induce her to take any exercise other than a rare and occasional ride in a bath chair. In no long time her dyspepsia was cured, her health restored, her heart rehabilitated, and her murmur gone, but no persuasion could induce her even for one instant to relax her watchful avoidance of every possible form of exertion. As may be imagined, she became ere long extremely obese, and after twenty years of positive inaction she died of apoplexy, having thrown away her life from a foolish faith in an idea of her own based upon no firmer foundation than a rash diagnosis. In the course of these lectures I have so repeatedly pointed out the variability of murmurs and their occasional cessation, that at first sight it seems scarcely worth while to say more upon the subject; but this is a matter upon which we can hardly say too much, as it is not only important to point out that murmurs occasionally vanish and are often variable, but also to show how a diagnosis may be made without placing any undue dependence upon either their presence or absence.

It must be remembered, then, that it is not the loudness of a murmur that indicates danger—the case is often quite the reverse; and it must also be remembered that a murmur may vanish under two very different conditions: first, because the heart becomes rehabilitated, the murmur ceases because its cause is gone; there is no longer any imperfection of the cardiac mechanism to produce it; there is no lesion to be revealed; and, second, the murmur may disappear, the lesion still remaining. Complete rehabilitation is a matter with



which the tricuspid valve only has been popularly credited among physicians, and this largely at the expense of its perfection; it has been credited with a safety-valve action which was supposed to enable it to open and shut according to the needs of the circulation. But indeed there is not one of the cardiac valves which may not be completely rehabilitated, after established incompetence, under certain circumstances and in certain conditions. In the lectures upon "Curable Mitral Regurgitation,"<sup>1</sup> and upon "Curable and Incurable Tricuspid Regurgitation,"<sup>2</sup> I have already entered fully into the various conditions under which the rehabilitation of these valves may occur. It is unnecessary therefore to say more upon this matter, except to reiterate the importance of having always in view in our treatment the possibility of restoring the function of those valves even when free regurgitation has been established through one or even through both of them. In time and with patience we shall be more often successful than we perhaps dare to hope. The permanence of this restoration is a different matter, and depends upon the age of the patient, his habits, the nature of his occupation, and the primary cause of the dilatation that has made these valves incompetent. So that the prognosis for such a patient, though hopeful in many cases, is by no means so in all. In regurgitation through the aortic valve the conditions are much less favourable for rehabilitation; but it sometimes happens, though the restoration is always less stable and less likely to be permanent, however complete it may seem to be for the time. An aortic regurgitant murmur often varies so greatly in intensity that at times unpractised ears, to which it was previously distinctly audible, fail to recognise it, while any abnormal sounds within the lungs may obscure it to even ears that have had greater experience. Diminished intensity of an aortic regurgitant murmur may be due to an alteration in the blood itself; more often to a lowering of the arterial blood-pressure,

<sup>1</sup> Lecture VI. p. 167.

<sup>2</sup> Lecture VII. p. 197.

and probably still more often to weakening of the cardiac systole. In the case of one very loud musical diastolic aortic murmur the patient never felt well unless his wife could hear his murmur across the dinner-table. Now and then we find a pathologist exhibiting the trickling of a small stream of water between the segments of an aortic valve, incompetence not having been recognised during life; it may not even have existed while the patient was alive, and any way the force employed in causing so trifling a stream most probably was quite insufficient to make it sonorous. Competence seems sometimes to be restored to the aortic valve by the deposition of fibrine upon the diseased segments;<sup>1</sup> in such a case the diastolic murmur gradually diminishes in intensity until it may entirely cease, the systolic portion of the murmur continuing audible, though even this may vary in pitch as well as in intensity. I myself have more than once had occasion to observe apparently complete rehabilitation of the aortic valve, under treatment, in cases in which regurgitation was due to separation of the segments from dilatation of the ascending aorta. In these cases the diastolic murmur died completely away under the use of iodide of potassium, which appears to have a twofold action; first it lowers the blood-pressure, and, second, it seems to have a contracting and thickening effect upon the coats of the arteries themselves. That the disappearance of the diastolic murmur was mainly due to the latter action seemed probable from its continued absence long after the drug had ceased to be given, the accentuated aortic second remaining.

Murmurs of regurgitation, may, as you know, occur at any of the cardiac orifices from dilatation of the parts with which the valves are connected, apart altogether from any alteration or disease of the valves themselves. It is under these circumstances that rehabilitation of an incompetent

<sup>1</sup> Of this Dr. Gairdner has recorded one case in the *British Medical Journal* for 30th March 1872, p. 334; and Dr. Walshe another in his *Diseases of the Heart*, 3rd edition, p. 386.

valve is most likely to take place, accompanied by perfect restoration of the normal sound, though—be it first or second—that sound may remain, for a time at least, accentuated. This is specially the case with the second sound, and from the prevailing conditions accentuation of it is probably permanent. With a first sound it is different; accentuation of it is not an uncommon sign of commencing dilatation of the ventricle, but as a dilated ventricle is capable of more perfect restoration than a dilated aorta, the sound itself after restoration becomes more nearly normal. Murmurs of obstruction belong to quite a different category; they invariably result from some direct and material obstacle to the onward current of the blood, and this obstruction is never the result of cardiac dilatation, though it is often its cause. The consequence of this is, that though murmurs of obstruction may vary considerably, or may even vanish, the lesion still remains, there is no change in the patient's condition, and the cardiac disease proceeds at an unaltered pace on its downward career. It is of importance to remember these facts in connection with the prognosis we may have to give the patient, or rather to his friends. In the one class of cases—vanishing of a murmur due to dilatation—the disappearance of the murmur may be the first indication of returning health. In the other class of cases—those in which the murmur has been due to obstruction—the vanishing of the murmur is merely an indication of some alteration in the composition of the blood, in the force of the cardiac systole, or in the physical relation of the parts, which may either interfere with the formation of the murmur, or hinder its propagation to the surface of the chest. It is not the first time that the disappearance of a murmur, in circumstances when this could not depend upon the cessation of its cause, has been hailed with undeserved delight as an indication of renewed life. Nay, a treatment has even been instituted which could have no other end than a simple masking of the affection, by influencing the physical causes

engaged in the production of the murmur. I refer to the method of treating cardiac disease by depletion and starvation, happily not much in vogue in the present day, but which played no unimportant part not so many years ago, and which is now being displaced by other equally unscientific and not less injurious methods.

In my last lecture I pointed out that a systolic murmur in the pulmonary artery depending upon abnormal alteration in the parts concerned, might disappear whenever the physical conditions were so changed as to render the production of a murmur impossible. When a pulmonary systolic murmur, however, depends upon actual physical obstruction, variations in the murmur are less likely to happen, and I have never observed any. A similar statement may be made in regard to a systolic aortic murmur dependent upon physical obstruction, and as this murmur depends either upon physical obstruction or upon aortic dilatation, an almost equally irremediable condition, I agree with a late writer on the subject that considerable variations are less frequently met with in murmurs of this character than in murmurs associated with affections of other valves.<sup>1</sup>

It is in connection with stenosis of the mitral opening that we find the greatest amount of variation prevalent. I have already entered pretty fully into this matter;<sup>2</sup> and in the wards of any hospital you may have daily opportunity of verifying the statements made, and of observing that mitral stenosis is at least as frequently associated with an entire absence of murmur, with an irregularly intermittent systolic mitral murmur, or with a more permanent systolic mitral murmur, as with the ordinary presystolic murmur with which it is popularly more immediately connected. The following case is a very interesting example of extreme variability in a murmur connected with this lesion.

<sup>1</sup> "On the Variation and Vanishing of Organic Valvular Murmurs," by W. R. Sanders, M.D., *Edinburgh Medical Journal* (January 1869), p. 584.

<sup>2</sup> Lectures IV. and V. pp. 117 and 135.



CASE XXVII. J. Munro, aged twenty-one, admitted to Bed No. 3 in Ward V. on 14th December 1870. This patient had been under my care for some time three years previously; at that time he had a well-marked presystolic murmur, but of so variable a character that it repeatedly vanished while being listened to. Under treatment he improved much in health, and after his discharge he came repeatedly under observation with a slightly hypertrophied heart, and an occasional presystolic murmur. He afterwards fell off greatly in health, and was readmitted with a feeble heart, very irritable, in an almost constant state of irregular palpitation, and with a permanent systolic murmur. He was presently able to be discharged somewhat improved, with the systolic murmur still persisting. At the above date he was again readmitted on account of catarrhal symptoms, aggravated by privation and want of shelter. After treatment for some time his murmur was found to have entirely vanished, there was some degree of cardiac hypertrophy, a prolonged first sound, and an accentuated pulmonary second, but nothing more. On 1st February 1871 he was shown to the Medico-Chirurgical Society without a trace of any murmur remaining. This case was all the more interesting and instructive in that, from circumstances connected with re-arrangement of the wards, he had been for some time under the care of Sir Thomas Grainger Stewart, who was able to testify as to the persistence of the mitral systolic murmur at the time of the patient's discharge from his ward, and to verify its complete disappearance when the patient was exhibited to the Medico-Chirurgical Society.<sup>1</sup>

Cardiac murmurs are thus, as I have just said, liable to very great variations, and may even vanish entirely beyond the ken of much more accomplished auscultators than that hypothetical individual, the average medical practitioner, the

<sup>1</sup> Vide *Edinburgh Medical Journal* (March 1871), p. 832. For ten years subsequently this patient continued with his cardiac condition unchanged; he was then lost sight of.

lesion still remaining. It is evident, therefore, how important it is to know how to determine in any given case—such as the hypothetical case I commenced with—the probable presence of a cardiac valvular lesion in the absence of any murmur; and this fortunately is not so impossible as at first sight it seems. From what has just been said, you will have learned that aortic systolic murmurs may vary considerably in intensity, but never disappear entirely; that aortic diastolic murmurs only vanish in the rarest instances, either spontaneously or under treatment, while they are always associated with a systolic murmur which persists and reveals the presence of the lesion. A systolic pulmonary murmur, if organic, is permanent; if non-organic it may vanish temporarily during deep inspiration, but it only vanishes permanently with its cause. A tricuspid murmur of regurgitation is most commonly temporary, and when it ceases to be heard, this may be accepted as an indication that the cause has ceased and that the heart has become rehabilitated. A tricuspid murmur of obstruction is rare, and those I have heard have neither varied nor vanished. The greatest interest connected with a vanishing murmur, therefore, centres in the mitral valve, the condition of which we have the most ample and varied means of ascertaining apart from the presence of a murmur. It is fortunate it is so, for so much discomfort sometimes centres round even its slighter ailments, that not long since I had occasion to see a gentleman, thus obscurely affected, who had travelled all the way from South America, only to obtain a definite opinion as to his exact organic condition, which was subject of dispute among his medical advisers in that far-off region.

The most distinct and permanent indication of organic lesion of the mitral valve is an accentuated pulmonary second sound. In the absence of any other cardiac lesion, or of pulmonary congestion from any other cause, persistent accentuation of the pulmonary second, distinctly recognised,

may be accepted as a certain indication of a lesion of the mitral valve. In one case<sup>1</sup> this accentuation of the pulmonary second was for long the only detectable indication of cardiac disease, and it was many weeks before we could obtain confirmative evidence by the detection of a mitral murmur, though we sought it daily. Such a state of matters is, however, comparatively rarely observed; more usually there are other indications which we only require to feel along the lines certainly to detect. As an indication of how this may be done, I shall give you a case which is not altogether hypothetical.<sup>2</sup> Suppose you have a case whose lower extremities are somewhat œdematous, both radial pulses equal, regular or possibly irregular, and distinct, but not jerking, even when the wrist is elevated. These facts indicate the probability of there being some affection of the heart, and the certainty if this is so that this affection is mitral and not aortic. The patient's face is ruddy; his hands and feet are chilly and of a purple colour marbled with red. These facts point to venous congestion with partial oxygenation of the blood through the skin; greater where that is fine, as on the cheeks; less so where that is thicker, as on the extremities. These signs indicate some hindrance to the circulation. On being stripped and laid down we find no undue arterial pulsation anywhere visible. The veins of the neck are not prominent; they exist but as thin blue lines; nevertheless on careful observation we find distinct pulsation in the jugulars. This points evidently to tricuspid regurgitation of some standing; this regurgitation, the venous remora, and the œdema might possibly be due to repeated attacks of bronchitis. On inquiry, however, the patient tells us that he has never had any bronchitis, and that he never had any cough till subsequent to an attack of acute rheumatism from

<sup>1</sup> Case of Elizabeth Baillie, aged nineteen, admitted to Ward XIII. 29th March 1873; discharged 31st May 1873.

<sup>2</sup> I here narrate a tolerably well-marked case; but a similar method of interrogation is applicable to all, the degree of certainty attainable varying with the distinctness of the facts.

which he suffered some four or five years previously; this naturally increases the probability that our surmise as to the existence of cardiac disease is correct. On placing a hand over the cardiac region we find two distinct centres of pulsation; the one occupies the lower end of the sternum, and there we have a diffuse, heaving, and somewhat forcible impulse; the other lies to the left of the sternum above the fourth rib, and there the impulse is less forcible and occupies less space, but is still diffuse; the apex beat is absent from its normal position, and it is not to be felt either below this or to its left. These facts further confirm the diagnosis of a cardiac lesion, and supply certain data upon which an opinion may be hazarded as to the exact nature of that lesion without having recourse either to percussion or auscultation; by these procedures we may indeed confirm our diagnosis, but the data they afford are not so reliable as those we have already ascertained. The heaving impulse at the lower end of the sternum indicates the existence of dilatation with hypertrophy of the right ventricle. The dilatation is confirmed by the jugular pulsation, as this proves long-continued regurgitation through the right auriculo-ventricular opening. Regurgitation through this opening might be due to stenosis with permanent patency. This, however, is a condition so rare that it may be practically set aside, and, besides, such a lesion has special signs of its own and is seldom found connected with so much hypertrophy as is present in this case. The dilated hypertrophy of the right ventricle is further proved by the absence of the apex beat from its normal position, the left ventricle taking no part in the tangible phenomena connected with the cardiac pulsation, as it is pushed backwards into the chest by the right ventricle lying like a water-cushion in front of it (*vide* p. 199). Further, the complete disappearance of the apex beat indicates that the left ventricle has no share in the dilated hypertrophy of the heart, even if it be not somewhat atrophied. But if the left ventricle is neither dilated nor hypertrophied, the left auricle is certainly both;



this is revealed by the distinct though not very forcible pulsation to the left of the sternum above the fourth rib, and if we can discover the cause of this dilatation we shall then get into our hands the end of the clue by which we may be guided through the labyrinth of all the sequential phenomena. Dilatation and hypertrophy of the right ventricle, apart from any pulmonary cause, which in this case does not exist, must depend on some obstruction to the onward flow of the blood within the heart itself. Obstruction at the entrance of the pulmonary veins into the auricle is unknown, and if it did exist would prevent the occurrence of dilated hypertrophy of the left auricle, which in this case is present. Aortic regurgitation has a peculiar pulse of its own, which in this case is not detectable; moreover, the consecutive changes which affect the left ventricle are also wanting. Aortic obstruction is incompetent to produce such an alteration on the left auricle as we have here; moreover, the changes which this lesion produces, if extreme, involve the left ventricle even more than the right, which is not the case here. *Per viam exclusionis*, we thus arrive at the mitral opening as the only possible position for an obstruction that could efficiently produce the phenomena observed. This obstruction can only be a permanently dilated state of the mitral opening, or a permanently constricted condition of it. In the former case regurgitation is the sole cause of obstruction, in the latter, regurgitation plays a very small part in preventing the onward flow of the blood. With a dilated opening the obstruction is only constructive, and arises from so much blood escaping backwards which ought to be thrown forwards. As regards the circulation the results are similar to those of aortic regurgitation (*vide* p. 75), though from the different position of the lesion these results act very differently on the heart itself. The regurgitation into the auricle, occurring during early diastole while that organ is filling from the lungs, tends undoubtedly to dilate that cavity; but this tendency to dilatation of the auricle is counteracted by the

patency of the pulmonary veins, as the regurgitant force is thus diffused over the whole vascular area of the lungs, instead of being concentrated on the auricle alone. Hence congestion of the lungs, of the right side of the heart, and of the general venous circulation, are early, common, and well-known results of mitral regurgitation, while great dilatation of the left auricle is in these circumstances almost unknown. There is no hindrance to the passage of the blood from the auricle to the ventricle; this indeed is even more easy than usual by reason of the wide and permanent patency of the auriculo-ventricular opening. There is thus neither accumulation within the auricle to dilate it, nor any need for increased driving force to hypertrophy it. It is quite otherwise in the case of stenosis of the auriculo-ventricular opening. In this case there is not only regurgitation through the narrow but permanently patent orifice in the valve which maintains the auricle and lungs in a constant state of repletion, but to this there is superadded a positive obstacle to the onward flow of the blood from the diminished size of the ventricular inlet. The concurrence of regurgitation with obstruction naturally produces a degree of congestion in the auriculo-pulmonary circuit, and a tendency to dilatation of the auricle, commensurate mainly with the degree of obstruction, as the one part of that circuit susceptible of this alteration. I need scarcely add that the necessity of forcing the blood through the constricted mitral opening causes the auricle to be always more or less hypertrophied. It is therefore as the result of constriction of the auriculo-ventricular opening that the auricle attains its greatest dimensions both as regards dilatation and hypertrophy, and these are rarely noteworthy at all except in those circumstances. There is thus little room for doubt that the considerable dilatation and hypertrophy of the left auricle in the case under consideration is due to actual disease of the mitral valve causing stenosis of the left auriculo-ventricular opening. Further, as the considerable change in the left auricle, and the great

consecutive alteration in the right ventricle, as well as in the circulation generally, have occupied but a short time in development—only five years having elapsed since the patient's first attack of rheumatism—the probability is that the stenosis of the mitral opening is considerable, and the prognosis is consequently grave. You see, then, that without putting the stethoscope on the chest an inquiry based upon Forget's law of retro-dilatation<sup>1</sup> has enabled us to make a distinct and most probable diagnosis, and to deduce an evidently accurate prognosis, while the same data would also enable us to propound a rational treatment. Could the use of the stethoscope enable us to do more? Certainly not. Could the stethoscope alone enable us to do so much? Not so readily as we are apt to suppose. If we had to trust to the stethoscope alone in such a case, what might we learn or not learn from it? We know that the murmur supposed to be distinctive of mitral regurgitation through a dilated auriculo-ventricular opening is a systolic murmur with its position of maximum intensity over the cardiac apex. And we also know that the murmur distinctive of stenosis of the mitral opening is the so-called presystolic murmur, which actually occupies the latter part of the diastole and runs up to and terminates in the first sound. But, as has been already pointed out (*vide* p. 247), a presystolic murmur is of all murmurs that most frequently absent, even when the physical condition of which it is distinctive is present. There are many cases of mitral stenosis in which the only murmur present is not presystolic but systolic in rhythm (*vide* p. 143), and very often there is no murmur at all (*vide* p. 252). If we trust, then, to auscultation alone; if we attempt to diagnose the exact nature of any cardiac lesion by careful consideration of the audible phenomena with due regard to their rhythm, and probable cause as suggested by their position of maximum intensity, without being actually

<sup>1</sup> *Précis Théorique et Pratique des Maladies du Cœur*, C. Forget, Strassbourg et Paris, 1851.

misled, we shall yet often fail of that accuracy of diagnosis which is always important and is generally quite attainable. There is no case of cardiac disease in which this is more likely to happen than one similar to that we have just been considering. We see, then, that in the case before us, as well as in similar cases, the stethoscope alone may fail to give us such distinct and positive information as will suffice to make an accurate diagnosis, and yet we are enabled to attain to this by a due attention to facts which were equally accessible to our forefathers in the prestethoscopic ages. These facts owe their significance to the modes in which each individual cardiac lesion is compensated; they vary naturally in degree according to the gravity of the lesion present; but their nature is always the same for similar lesions, presenting in this respect a marked and important contrast to the acoustic phenomena only too often implicitly relied upon. These acoustic phenomena vary not only in degree, but are often loud and well marked when no important lesion exists, and not uncommonly absent when serious lesions are present.

In connection with this subject I may recall to your memory the statement by Bellingham<sup>1</sup> that in certain conditions there may be developed a diastolic murmur of aortic origin, unaccompanied by any lesion of the aortic valve or by aneurysm. A case of this kind has been recorded by Professor Law,<sup>2</sup> and another by Dr. Finlayson<sup>3</sup> of Glasgow. Like Dr. Hayden<sup>4</sup> I have never seen such a case, and have also a difficulty in understanding Bellingham's explanation of the mechanism by which he supposed the diastolic portion of this murmur to be produced.

A careful attention to the rhythm and position of maximum intensity of murmurs, and the same care directed to the physio-pathology of the heart where no murmurs are present, will enable us to unravel these difficult

<sup>1</sup> *A Treatise on Diseases of the Heart* (Dublin, 1857), p. 152.

<sup>2</sup> *Pathological Society's Reports*, vol. iii. p. 3, March 1868.

<sup>3</sup> *Lancet* (1885), vol. i. p. 426.

<sup>4</sup> *Op. cit.* p. 845.



and embarrassing cases with a skill proportioned to our experience.

Morbid anatomy reveals and describes a diseased structure; pathology unravels the process by which that structure has become diseased. What I wish to be understood by the term physio-pathology or pathological physiology is a study of the consecutive changes which occur, not as part of a morbid process, but which are developed as compensatory to that process. Thus rheumatism may shrivel the aortic valve, but it does not hypertrophy the left ventricle. This is a purely physiological development induced by the necessity of compensating changes in the physiological relations of the parts caused by a pathological alteration of their structure. This affords therefore an apt illustration of what I wish to be understood by the term physio-pathology of the heart, of which this hypertrophy and the other relative changes constitute one phase.

While freely acknowledging the importance of the stethoscope in the diagnosis of cardiac disease, I would have it remembered that it is not all-sufficient, and that in order successfully to interpret all its revelations we require to pay careful attention to the condition of the heart itself, always remembering the consecutive manner in which those alterations occur, which never fail to follow any special lesion. In many cases a knowledge of these consecutive changes will enable us to form a correct diagnosis apart altogether from any acoustic phenomena, which may or may not be present. By feeling along the lines indicated by our knowledge of physio-pathology, we shall always be able to give a rational account of our case, both in regard to diagnosis and prognosis, though it may not always be so easily unravelled as the one we have just been considering.

## LECTURE X

### ON INTERMITTENCY AND IRREGULARITY OF THE PULSE; PALPITATION, CARDIAC AND AORTIC; TREMOR CORDIS; TACHYCARDIA; BRADYCARDIA; AND DELIRIUM CORDIS

THERE is perhaps no single symptom connected with the heart which gives rise to so much discomfort, uneasiness, and feeling of insecurity as intermittency or irregularity of the pulse. Yet these symptoms are quite as often found apart from any serious disease of the heart as in connection with it.

The heart, we know, beats because its muscular fibre is incompletely differentiated, and still retains the power of spontaneous movement possessed by all primordial protoplasm.<sup>1</sup> The heart's energy resides in its muscular fibre, and its quality depends upon the perfection of the cardiac metabolism.

The nervous system neither initiates nor maintains the rhythmic movements of the heart, but it controls and regulates them, and through it these movements may be variously modified or even arrested.

The surface of the heart, particularly at its base, is covered by a network of nervous filaments, and these are connected on the one hand with various ganglia scattered throughout the substance of the heart, particularly at the junction of the *sinus venosus* with the auricle, and in the auriculo-ventricular sulcus. On the other hand these filaments unite in forming three distinct strands or nervous cords by which they are

<sup>1</sup> Foster's *Textbook of Physiology*, 5th edition (1888), p. 288 *et antea*.

united to the central nervous system. One of these cords (H, Fig. 16) passes through the first dorsal and the last cervical ganglion into the sympathetic nerve, and through it there pass to the heart those impulses which increase the rate of its pulsations and augment their force.<sup>1</sup> This is the *katabolic* nerve of the heart,<sup>2</sup> by its action cardiac metabolism is effected and the heart's energy set free; but we must remember that its action is twofold, and that these actions

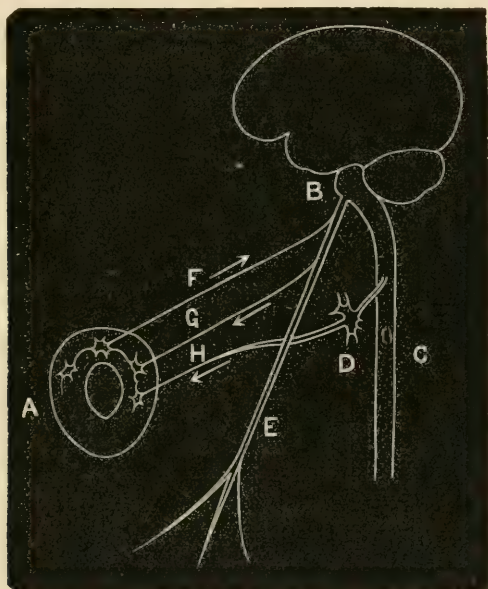


FIG. 16.

do not always coincide, thus by it both the rate and force of the heart's pulsations may be simultaneously increased, and we may have a heart-beat with a quick, large, and full pulse, or we may have only a rapid heart-beat, the pulse remaining small and empty.<sup>3</sup>

<sup>1</sup> *Untersuchungen über die Innervation des Herzens*, von Albert v. Bezold (Leipzig, 1863), Erste Abtheilung, S. 162.

<sup>2</sup> Gaskell, *Journal of Physiology*, vol. vii. pp. 41 and 46.

<sup>3</sup> "Sometimes the one result and sometimes the other being the most prominent," Foster, *op. cit.* p. 294; *vide* also Roy and Adami, *Transactions of the Royal Society*, vol. clxxxiii. p. 240.

The inferior cardiac nerve, the second of the cords connecting the heart with the central nervous system, is the *anabolic* nerve of the heart (G, Fig. 16), and its action is to inhibit the action of the augmentor or katabolic nerve.

The third nervous strand is the superior cardiac nerve (F, Fig. 16); this conveys from the heart to the vaso-motor centre in the *medulla oblongata* an influence which enables it to regulate and control the movements of the arterioles in accordance with the needs of the heart. When the blood-pressure is too high and threatens to overpower the heart, an impulse sent through this nerve inhibits the constrictor influences and tempers down the blood-pressure to suit the cardiac strength.<sup>1</sup> This nerve is often called the depressor nerve of the heart.

The inferior and superior cardiac nerves both enter the trunk of the vagus nerve and ascend to the brain along with it, but each has its separate origin as well as its special function. The inferior cardiac nerve is generally regarded as a branch of the vagus nerve, and its action is referred to as vagus action, but it has a special root of its own, and in that, as well as in action, it is much more closely connected with the spinal accessory nerve than with the vagus proper.

You will remember, then, that the superior cardiac nerve controls the blood-pressure in the interests of the heart through the vaso-motor centre in the *medulla oblongata*.

The inferior cardiac, anabolic, or vagus nerve, when in action, counteracts and inhibits the action of the katabolic nerve, slows the heart generally, lessens the excitability of the ventricles, and may even when acting moderately reduce their output by as much as thirty per cent,<sup>2</sup> thus initiating residual accumulation and all the evils that may flow from it.

On the other hand the katabolic nerve, though it does not initiate or cause cardiac movement, when in action accelerates and augments it, and is itself kept in check by the

<sup>1</sup> Foster, *op. cit.* p. 351.

<sup>2</sup> Roy and Adams, *op. cit.* p. 217.



anabolic nerve. It is closely connected with the sympathetic system.

The natural tonicity of the heart is the property of the cardiac muscular fibre, and depends upon the perfection of its metabolism; the heart's pulsation is also muscular and automatic, but its rate, rhythm, and force are controlled by the interaction of the nerves just described acting under the influence of various excitants.

The anabolic or vagus nerve is excited to action by inhibitory influences that pass to it from every quarter through the cardio-inhibitory centre. An inhibitory influence may either be physical or psychical; it may come from a diseased organ, a depraved secretion, or a mental shock. Under any such influence the vagus nerve may slow and reduce the action of the auricles, and may even arrest it for hours. The action on the ventricles is similar, but not so powerful; strong stimulation of the vagus may indeed arrest the action of the ventricles, but never for a period long enough to endanger life. At a certain point of vagus inhibition, that varies in different animals, the ventricles commence to beat independently of both sinus and auricles, and this idio-ventricular action, which is at first slow and irregular, gradually attains a reasonably rapid rate and becomes almost perfectly regular in rhythm. Irregularity is brought about by the auricles failing to respond to some of the contractile impulses which reach them from the sinus, or by the interference of the sinus and the ventricular rhythms with each other, which is a usual cause of this abnormality.<sup>1</sup> Ultimately the cause of all irregularity may thus be traced back to inhibition of the heart through—but not by—the vagus.

The effects of vagus inhibition vary from simple intermission to sudden death, and between these extremes we have irregularity which is almost invariably accompanied by a certain amount of dilatation. Therein constitutes its danger, a danger which after middle life is far from being

<sup>1</sup> Roy and Adami, *op. cit.* pp. 293, 294, etc.

hypothetical. So long as the pulsation of the heart is purely automatic, that is so long as the foetus remains in utero, intermittence seems to be unknown; no doubt there are agencies even then which ought to give rise to intermittence, but if this ever occurs, it must be very exceptional, as I am not aware of a single case that has been recorded. It is otherwise after birth, then injurious agencies multiply so that infantile intermittence is a well-known fact. From man's birth to his grave intermission is a matter of common occurrence, and, as mere occasional intermittence, it does not seem to have any marked injurious effect. It always, however, handicaps a man more or less, and for his own peace of mind it is desirable to get rid of it, quite apart from the fact that even simple intermission always indicates a feeble myocardium, and if neglected may prove the precursor of more serious ailment.<sup>1</sup>

Simple intermittence is always an early intimation of cardiac failure due to anæmia, overwork, or worry; it may be associated with valvular lesion, but has only an incidental connection with it. The more feeble the myocardium the more readily intermittence may be brought about, but it seems as if even an average heart might be forced to intermit if the exciting cause be powerful enough. Thus I have known a young man with an apparently strong heart forced to intermit by the shock of a railway accident. In this case the intermissions were at first every second beat, but in a few months they died down to one intermission in twenty beats, and I have no doubt they ultimately ceased entirely. Sir Benjamin W. Richardson has recorded a case of shipwreck in which the fear of instant death from drowning caused the heart of a middle-aged man, in perfect health and spirits, suddenly to stop. He was rescued and put on board another vessel, and when he had regained sufficient composure he found that his heart intermitted four or five times a minute. At first these intermissions were so disturbing as

<sup>1</sup> Vide *The Senile Heart*, p. 41, etc.

to prevent sleep; by and by they died away to two in a minute, and the patient was no longer cognisant of them unless he felt his pulse.<sup>1</sup> In another case also related by Richardson, mental anxiety alone developed persistent intermittency, followed by death from the silent but sleepless suffering induced,<sup>2</sup> complicated, no doubt, by sequential dilatation of the heart.

Emotional inhibition of the heart is often a most serious matter, and may instantly and fatally arrest its action. This has happened often enough in those not known to labour under any cardiac affection, to make it a possible occurrence in any one with a myocardium weakened by age, or from some definite cause at an earlier period of life. Zimmermann<sup>3</sup> has published a number of cases of sudden death from cardiac inhibition, and from him we learn, what we would scarcely have expected, that unexpected joy is even more dangerous to life than sudden grief.

Sophocles at the age of ninety died suddenly of joy on being crowned as the first tragic poet of the age. Philpides, the comic writer, died a similar death. Chilon of Lacedæmon died in the arms of his son who had borne away the prize at the Olympic games. The famous Fouquet died of joy on being set free by Louis XIV. The niece of Leibnitz died suddenly of joy at finding a box containing ninety thousand ducats beneath the philosopher's bed. Many other similar cases have been recorded both by Zimmermann and others, all testifying to the fatal effect of excessive emotion even when of a pleasing character. It seems more natural that terror and grief should be more hurtful than joy, and though this does not appear to be the case, yet these emotions have in their turn been fatal to many. Philip II., king of Spain, enjoys the unenviable notoriety of having frightened two of his counsellors to death. One of his ministers of state died

<sup>1</sup> *Transactions of the St. Andrews Medical Graduates Association* (1870), p. 238.

<sup>2</sup> *Op. cit.* p. 239.

<sup>3</sup> *A Treatise on Experience in Physic* (London, 1782), vol. ii. p. 286.

suddenly on being sharply rebuked for a hesitating answer. Another, the Cardinal Espinoza, died a few days after being sternly told, "Cardinal, know that I am master." Mallet's tale of "Edwin and Emma" was founded upon an actual incident that happened at Bowes in Yorkshire, in 1714, when a young woman died suddenly on being told of the death of her lover. Palmer, a celebrated comedian of last century, died on the stage of the Liverpool theatre in most tragic circumstances. Palmer had recently lost both his wife and a favourite son; he was acting the part of "The Stranger" in the play of that name, when one of the *dramatis personæ* in the course of the play asked him for his wife and family. Palmer, unable to reply, became inexpressibly agitated and dropped dead upon the stage. In the end of last century Prince George Louis of Holstein, having removed the body of his wife from one coffin to another of more costly materials, desired his valet to read him some pages from a pious book, and kneeling at the side of the coffin he burst into tears and died. And a few years ago there occurred in France an even more startling instance of the fatal effect of overwhelming emotion. Dr. Deleau, a celebrated aurist, only forty-four years of age, leaning over his dying daughter to receive her last farewell, himself fell dead as if struck by lightning.

The effects of emotional inhibition vary with the intensity of the emotion on the one hand, and the sensitiveness of the organism on the other, to which we must add a third and most important element, the condition of the heart itself. In some, from an untoward combination of these conditions, the emotion may prove suddenly fatal; in others the disturbed rhythm of the heart may persist as a fact of which the sufferer cannot for one instant lose the consciousness, and which as a natural sequence terminates in dilatation of the heart with death looming in the distance. On the other hand when the conditions are more favourable, the effect dies off with the fading of the emotion which produced it. There are probably but few of us who have not at one time



or other realised in ourselves the meaning of an emotional inhibition when our heart has stood still, as the expression is, in the face of some impending danger to ourselves or others. It has been graphically referred to by our vernacular poet Ballantyne as an indication of maternal anxiety :

“ My vera heart gaes loup, loup,  
Fifty times a day.”

The “ loup ” being nothing but the perceptible thump, the forcible systole, which succeeds a momentary intermission (inhibition). Emotion acting in this way is reflected from the sensorium to the cardio-inhibitory centre in the *medulla oblongata*, and from it to the heart through the inferior cardiac branch of the vagus.

But while psychical emotion may be so disturbing to the heart and so disastrous to the individual, any physical sensation of equivalent intensity may be no less injurious both to the organ and the organism. Severe cold applied to the surface has been no infrequent cause of fatal inhibition of the heart, especially in those exhausted by fatigue. During the Russian campaign (1812-13) the French soldiers were often seen to fall dead as if struck by lightning from the effects of the excessive cold. At Smolensko more than thirty grenadiers of the Italian Guard fell dead from this cause, while attempting to set themselves in line on the height beyond the Borysthenes.<sup>1</sup> A cold bath has proved fatal to infants in this way, and many a one has died in the water from an inhibited heart. These unfortunates are said to be seized with cramp and to drown ; but the cramp is that of the heart fatally inhibited. Now and then such victims do struggle, and are partly drowned, for inhibitory paralysis of the heart, like every other form of asystole, is not necessarily instantaneously complete, but may be ingravescent in character. Cold applied internally is also

<sup>1</sup> *A Treatise on the Effects and Properties of Cold*, by Moricheau Beaupré, M.D., translated by John Glendinning, A.B. and M.D. (Edinburgh, Maclachlan and Stewart, 1826), p. 149.

well known as a frequent cause of fatal cardiac inhibition. The knowledge of this is of very ancient date indeed, for Quintus Curtius tells us that Alexander the Great lost more men from his soldiers drinking the ice-cold waters of the Oxus, than he did in any one of his many serious battles. Almost every summer some sudden death in the harvest field, from a similar cause, supplies a fresh modern illustration of the sudden way in which life may be brought to a close by a shock thus applied. Not cold only but pain also, or, in fact, any physical irritation, such as those arising from acidity, flatulence, undigested food, or any depraved secretion, as well as many poisons, such as tobacco, alcohol, etc., used in excess, may give rise to simple intermittence, or to various forms of irregularity, and ultimately to dilatation of the heart.

One hundred years ago the pathology of such cases was very much based upon Tristram Shandy's idea that the body and soul are like a coat and its lining; if you rumple the one you rumple the other also. The intensity of any impression psychical or physical constituted its danger. "A passion," says Zimmermann, "without even being carried to excess, will sometimes occasion a difficulty of breathing, together with a sense of stricture in the breast, and an hesitation to speak, the tongue remaining as it were immovably fixed to the palate. The weaker passions speak, the stronger passions are mute."<sup>1</sup> Great intensity of emotion was supposed to collapse the heart, so that it neither received nor emitted any blood, the victim dying instantaneously.<sup>2</sup> It was also known that the heart occasionally ruptured from the strain thus thrown upon it, as in the case of Philip V. of Spain.<sup>3</sup> Essentially popular in character, this pathology was of very ancient date, and when Shakespeare makes Malcolm say to Macduff:

"Give sorrow words; the grief that does not speak  
Whispers the o'erfraught heart and bids it break,"

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<sup>1</sup> *Op. cit.* p. 267.

<sup>2</sup> *Loc. cit.*

<sup>3</sup> Zimmermann, *op. cit.* p. 274.

he but expresses in terse poetic form the weak periphrasis of Zimmermann.

Since the beginning of this century men have ceased to be contented with vague descriptions of still vaguer ideas, but have sought to connect physical results with definite physical causes. Among the earliest pioneers in regard to the influence of injuries of the nervous system on the heart, were Legallois<sup>1</sup> and Wilson Philip.<sup>2</sup> Yet though the pathological acumen of Alison<sup>3</sup> distinctly recognised the bearing of such experiments on the cardiac depression so often observed in the early stages of abdominal inflammation, as well as their bearing on the sudden death that results from a blow on the epigastrium, we had got no farther fifty years ago than to be able to connect fatal cardiac failure with concussion of some part of the cerebro-spinal or sympathetic system. The history of the last fifty years contains the history of the inquiry by which we have been led to recognise the method by which the heart's action is influenced by extraneous impressions. This knowledge not only helps our diagnosis, but it also directs our treatment and enables us to bring it to a successful termination, not only aiding us in relieving suffering of a most wearing character, but enabling us to restore to comfort and health hearts that have already commenced their downward career. The story of this progress is to be found in the writings of the brothers Weber,<sup>4</sup> of Budge,<sup>5</sup> Cyon,<sup>6</sup> Von Bezold,<sup>7</sup> and Rutherford,<sup>8</sup> writings

<sup>1</sup> *Expérience sur la Principe de la Vie, notamment sur celui des mouvements du Cœur et sur le siège de ce Principe*, paper read before the Institute of France, 3rd June 1811.—Vide *Œuvres de César Legallois* (Paris, 1830).

<sup>2</sup> *Philosophical Transactions*, 1815 and 1817; vide also *An Experimental Inquiry into the Laws of the Vital Functions* (London, 1818), 2nd edition.

<sup>3</sup> *Outlines of Pathology and Practice of Medicine* (Edinburgh, 1844), p. 12.

<sup>4</sup> *Omodei annali universali di medicina*, cxvi. pp. 225-233 (1845).

<sup>5</sup> *Archiv f. Anat. u. Physiol.* (1846), S. 295.

<sup>6</sup> *Bericht d. Sächs. Ges. d. Wiss.* (1866), S. 308.

<sup>7</sup> *Untersuchungen über die Innervation des Herzens* (Leipzig, 1863); vide also *Untersuchungen aus dem physiologischen Laboratorium in Würzburg* (Leipzig, 1867).

<sup>8</sup> Papers read before the Royal Society of Edinburgh, May 1869, published in abstract in the *Journal of Anatomy and Physiology*, vol. iii. (1869), p. 462,

which have culminated in the important work of Roy and Adami.<sup>1</sup>

An intermittent or irregular heart, and the one may be regarded as merely an advanced stage of the other, presupposes an enfeebled myocardium and an adequate excitation of psychical or physical origin, the stimulus acting on the heart through the inhibitory centre in the *medulla oblongata* and the vagus nerve. The more perfect the cardiac metabolism the more powerful must be the inhibitory impulse to produce any result, hence whatever interferes with cardiac metabolism conduces to irregularity,<sup>2</sup> and we must never forget that irregularity diminishes the efficiency of the heart without reducing its expenditure of energy.<sup>3</sup> Irregularity thus contributes to cardiac dilatation in a twofold manner; the vagus inhibition to which it is due diminishes the ventricular output,<sup>4</sup> and as this diminished output is accompanied by no diminution in the expenditure of energy cardiac metabolism is of necessity still further impaired.

I have occasionally observed a heart small from birth or from early childhood, but apparently perfect in its metabolism, to have, as age advanced, irregularity of various kinds forced upon it. In such a case we may have intermission, either only occasional or every second or third beat for days, or with occasional intervals for years; or the pulse may be irregular in both rate and rhythm for varying periods; or the heart may have a sudden fit of tremor, or two or three forcible systoles may be followed by a reversion to the normal rate and force. Such irregularities are most commonly due to gastric or intestinal troubles, but psychical emotions are equally efficacious in producing them. A small but otherwise healthy heart may suffer in this way for many years without any apparent detriment, certainly with much

and in full in the *Transactions of the Royal Society of Edinburgh*, vol. xxvi. (1872), p. 107.

<sup>1</sup> *Philosophical Transactions*, vol. clxxxiii. (1892), B. pp. 199-298.

<sup>2</sup> Von Bezold, *op. cit.* S. 279.

<sup>3</sup> Roy and Adami, *op. cit.* p. 284.

<sup>4</sup> *Op. cit.* p. 217.



less detriment than befalls a heart similarly affected for the first time after middle life.

Irregularity is only associated with valvular lesions incidentally; that is to say, no valvular lesion in itself has any special influence in inducing it, except in so far as from its own intrinsic character, or from its effect on the circulation, it has influenced the metabolism of the myocardium. Hence irregularity is a most characteristic symptom of mitral stenosis (*vide antea*, p. 124, note), and of cardiac dilatation, chiefly in its later stages or at any period if dilatation predominates over the hypertrophy which always accompanies it; theoretically we must also regard it as a symptom of atheromatous coronaries, but I confess never to have had unequivocal proof of this. Associated with any other valvular lesion irregularity must always be regarded as a symptom of cardiac failure.

The following sphygmogram (Fig. 17) represents the feeble irregular pulse of a patient with a perfectly healthy heart, whose life was rendered miserable by a feeble irregular pulse due to impaired metabolism of his myocardium, the result of a depraved state of his general health due to confinement during business hours to an unhealthy and badly ventilated office. A month's holiday with tonic treatment and fresh air speedily restored this patient to health.



FIG. 17.

The following sphygmogram (Fig. 18) represents the irregular pulse of mitral stenosis; in it you will observe that the ventricular output (as indicated by the line of ascent), though not so meagre as in the preceding case, yet maintains

a uniform low level, not so much because the ventricular output itself is diminished as because the ventricular income is persistently at a low ebb. In this matter it contrasts very strongly with the annexed sphygmogram (Fig. 19) of the pulse of a large dilated heart, in which small systolic waves are varied every now and then by a large one. The tracing shows a pulse irregular as to rate, rhythm, and also apparently as to force, but this is doubtful, as the occasional large ventricular output is more probably due to the accidental coincidence of the auricular systolic wave with that of idio-ventricular action.

In the treatment of intermission and irregularity we have always to deal with a myocardium irritable because

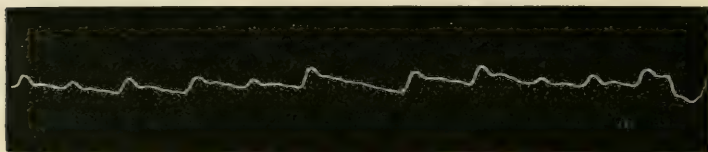


FIG. 18.

ill-fed, and sometimes, as in the case of a dilated heart, our principal treatment must be directed to the heart itself. On a future occasion I shall describe to you what cardiac tonics are, and how they ought to be employed. Strict and careful attention to diet is also requisite in all cases of cardiac irregularity, but as this does not differ from what is always required in all cases of heart disease, this also will be best considered when speaking of the general treatment of such cases (*vide postea*, Lecture XIV.)

There is another form of irregularity observed in various circumstances, and which is not uncommon when digitalis has been given for some considerable time: this is the *pulsus bigeminus* of which the accompanying sphygmogram (Fig. 20) gives a very good example. This tracing was taken from the radial artery of a man suffering from aortic incompetence. Traube gave this "twin pulse" a somewhat unenviable

notoriety by ascribing to it an important prognostic value as presaging a speedy lethal termination to the disease in which it showed itself.<sup>1</sup> Later observations have shown that this idea was not well founded, and cases in which it has occurred have been often enough known to end in recovery; it is merely a form of irregularity of no special importance.

Sphygmograms 21 and 22 represent the usual pulse and heart-beat in a case which frequently presented a very unusual form of irregularity seen in the annexed sphygmograms, of which Fig. 23 shows the heart-beat and Fig. 24 the pulse. The pulse-tracing shows that not unusual form of irregularity known as hemi-systolic bradycardia, of which I shall speak later, but the cardiogram shows that though the pulse was a bradycardiac pulse of the hemi-systolic type, the heart beating at the rate of 88 per minute and the pulse at only 44, the heart-beat itself was also bigeminal. You will observe in the cardiogram that the second of each twin beat is imperfect; it is less than half the amplitude of the first beat; its pulse failed to reach the radial artery and hence the bradycardia. But the bigeminal character of the heart-beat has impressed its own peculiarity upon the pulse tracing, which naturally differs somewhat from a hemi-systolic bradycardia of the ordinary type. Further, this heart presented a well-marked example of the *pulsus alternans* in that, although both ventricles beat simultaneously, the left and right ventricle pre-



Fig. 19.

<sup>1</sup> *Gesammelte Beiträge*, and *Berliner klinische Wochenschrift*, 1872, "Ein Fall Pulsus bigeminus nebst Bemerkungen."

dominated alternately. The heart beat bigeminally, and the two ventricles alternated in force. As there was free regurgitation with jugular pulsation, this alternation in force was well seen in the neck, where the carotid artery and

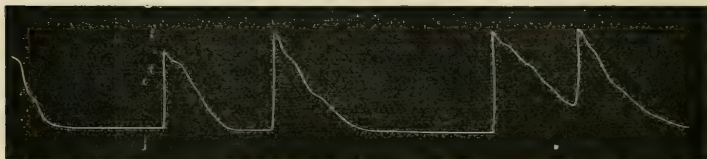


FIG. 20.

jugular vein were seen to beat alternately. This heart thus, and another that occurred about the same time, presented the rare combination of a bigeminal alternating heart-beat with

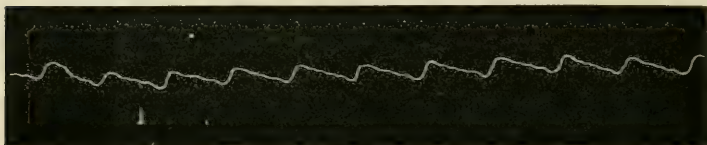


FIG. 21.

a hemi-systolic bradycardiac radial pulse also of a bigeminal type, forming altogether a very remarkable combination of irregularities.<sup>1</sup>

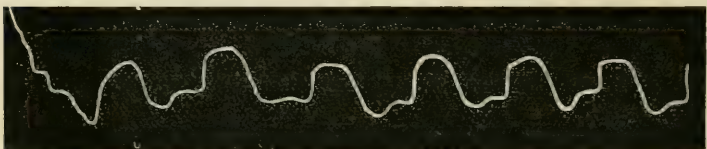


FIG. 22.

All who suffer or who think they suffer from disease of the heart are prone to complain of palpitation. But this term is loosely applied to every variety of cardiac abnormality. In true palpitation there is no irregularity; there is nothing

<sup>1</sup> *Vide* On two Heart Cases which presented a rare form of Irregularity, from Dr. G. W. Balfour's "Clinique," by Charles S. Roy, M.B., *Edinburgh Medical Journal* (1878), p. 594.



but rapid, regular, and violent pulsation of the heart, which often shakes the whole body and makes itself unpleasantly felt by the sufferer, accompanied by violent throbbing of the aorta, carotids, and larger arteries, but does not extend to the smaller arteries, the radial pulse for instance being rapid, but having no excess of force. These attacks come on suddenly, and last from a few minutes to many hours; they are extremely

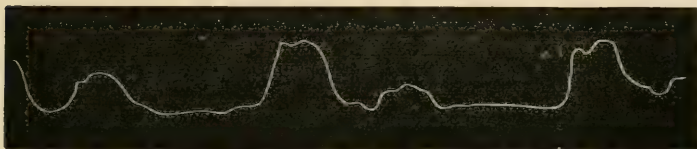


FIG. 23.

distressing and even alarming to the patient, but they are not usually attended by any danger. Palpitation is due to reflex inhibition of the inhibitory centre, which temporarily removes the vagus influence, and allows the heart to be run off with by the katabolic nerve acting as an accelerator only. Palpitation occurs in weakly, anæmic individuals, and is induced by psychical and gastric reflexes of various origins, never by exercise. The rapid, forcible augmentor action that follows



FIG. 24.

exertion in anæmic individuals is accompanied by a full and forcible radial pulse, while the heart-beat is never so violent and throbbing as in true palpitation, and both cease at once whenever the patient becomes quiescent. The rapid action that occurs in alcoholic and tobacco poisoning is a tachycardia (*q.v.*) and not a true palpitation.

Palpitation is so often of psychical origin that our most efficient cure is often the narcotic needle, and whatever its origin large doses of the bromides will frequently be found

our most useful adjuvant. Neurasthenia—imperfect metabolism—is at the bottom of all repeated or long-continued attacks of palpitation, and all our endeavours must be directed to the improvement of the general health. In some cases massage and forced feeding work a rapid cure; in others fresh air and exercise are of the greatest advantage. Formerly horse exercise used to be greatly recommended; in the present day no exercise is preferable to the moderate and prudent use of a bicycle, but no racing, nor pushing up hills, both of which are fraught with danger to a feeble heart. In recommending cycling for a weak heart, I necessarily presume that the heart is young and free from organic mischief; it is most unsafe for elderly hearts, or those with valvular lesions. Naturally various tonics, such as iron, arsenic, and strychnine, may be advantageously combined with the tonic regimen, every possible means being taken to promote a healthy hæmogenesis, and every endeavour made to put a stop to any undue hæmolysis.

There is a peculiar disease often complained of as palpitation, which has, however, a totally different syndrome. There is rapid action indeed, the heart beating 140 or more per minute, and the heart's action is violent, but there the resemblance ends, for in *Graves' disease*, of which I speak, the disagreeable throbbing is not confined to the heart and large arteries but extends over the whole arterial system. Add to this that in all such cases the heart sounds are loud and distinct, so much so that Graves, to whom we owe the earliest description of the disease, says in reference to one of his cases, "I could distinctly hear the heart beating when my ear was distant at least four feet from the chest."<sup>1</sup> The phenomena distinctive of Graves' disease are violent perturbative action of the heart and arteries associated with enlargement of the thyroid gland (goitre) and protuberant eyeballs (exophthalmos). In marked cases all three symptoms

<sup>1</sup> *A System of Clinical Medicine*, by Robert James Graves, M.D. (Dublin, 1843), p. 674.

are present, but often there are only two, and sometimes the violent throbbing of the heart and arteries is the only symptom present, but this is distinctive enough and quite sufficient to enable us to differentiate it from every other form of so-called palpitation. At first there is no change in the heart itself, but the rapid perturbed action is associated with diminution of the ventricular output, and this ultimately leads to residual dilatation with all its usual results, including albuminuria.

For this mysterious complaint many various remedies have been suggested and employed. All the elements of the tonic regimen have been more or less successfully employed, in combination with iron, arsenic, and digitalis. Baths of compressed air have also been used with benefit, and by some good results have been said to be attained by the use of thyroid extract, probably thyrocol—the active colloid substance of the thyroid gland might be used with better results. I myself have used thymus gland with excellent results, thyroid extract being too often found to increase the symptoms it is employed to cure. We cannot say that the thymus gland is antagonistic to the thyroid, but at least it atrophies as the thyroid grows, and the idea is thus suggested that there is some contrarious relation between their actions.

*Tachycardia* is another form of rapid heart action often confounded with palpitation, from which it differs *toto celo*. It is an error to apply the term tachycardia to all cases in which there is a rapid heart. For example nothing could be more striking than the contrast between the syndrome of Graves' disease and that of tachycardia: in both the heart hurry may last for years, but in the one case the heart's action is violent, perturbed, and noisy, and the whole arterial system throbs disagreeably; in the other the heart is rapid enough but feeble, and its sounds empty, like the tic-tac of the foetal heart, while the pulse, though quick, is feeble and sometimes almost imperceptible. One remarkable characteristic of tachycardia is the little disturbance it gives the

sufferer, who will often be found going composedly about his ordinary occupations with a pulse-rate of 150 or more. Donders was the first to point out that a rapid heart-beat

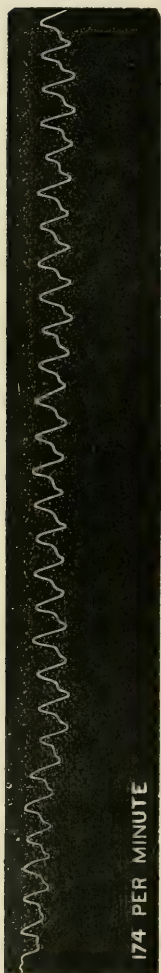


FIG. 25.

meant shortening of the systole and a diminution of the ventricular output,<sup>1</sup> hence shortening of the primary wave in the pulse-tracing and increased depth of the dirotic notch, dirotism of the pulse and pulse-tracing. When the pulse-rate is much increased, the pulse becomes hyperdirotic; the ordinary dirotic notch is carried on to the ascending limb of the tracing, and simulates anacrotism, as in the sphygmogram here given (Fig. 25). This is the tracing of a tachycardiac pulse, hyperdirotous, of low tension, beating perfectly regularly at the rate of 174 per minute, as counted on the sphygmogram. There was in this case increased præcordial dulness, and a feeble wobbling heart-beat, a condition obviously not without danger, the degree of which may to some extent be gauged by the amount of dirotism present, as this indicates diminution of the ventricular output (lessening of the heart's contraction volume), and tendency to death from sudden or ingravescient asystole. In tachycardia the pulse-rate is said often to exceed 200 per minute; I myself am quite unable to count a pulse beating over 150; by the aid of the sphygmograph we may

certainly count more, but I have never found the rate up to 200.

At both extremes of life we may have a rapid pulse; in infancy this depends upon the low blood-pressure and concurs

<sup>1</sup> *Nederl. Archiv voor Genees-en Naturk.* Bd. 11 (1865), S. 184.



with it in promoting the diffusion of the blood plasma and the rapid growth of the tissues, and it ceases as the blood-pressure rises and development takes the place of growth. At the other extreme of life a rapid, feeble pulse, weak impulse, and empty heart sounds, often indicate failure of cardiac energy and herald the approach of death. These two instances may be reckoned as forms of normal or physiological tachycardia, as may also the heart hurry that not infrequently accompanies convalescence from febrile diseases, the rapid pulse of some women during menstruation, and also during the puerperium. Tachycardia is not infrequently induced by vagus inhibition due to the immoderate use of tobacco, alcohol, or other poisons. In the case of alcohol the vagus inhibition is occasionally associated with neuritis, and with fibro-fatty degeneration of the myocardium, and this combination constitutes the chief source of danger in delirium tremens, and also in chronic alcoholism.<sup>1</sup> Tachycardia is also not infrequently the sole indication of the chronic valvulitis that sometimes follows heart strain and terminates in stenosis of the mitral opening. It may also be due to interference with vagus action by the direct pressure of an intra-thoracic tumour, or to reflex inhibition of the vagus by physical or psychical causes of various origins, sometimes taking the place of simple irregularity under conditions the exact nature of which are as yet unknown.

When the rapid heart depends upon recent strain of the myocardium the most hopeful treatment seems to be to maintain for some time the full action of belladonna (or atropine), as indicated by slight dilatation of the pupil. In other circumstances, remove the cause if this be at all possible, improve the metabolism of the myocardium by appropriate tonics, and quiet the action of the inhibitory centre by suitable sedatives properly employed. The condition is not devoid of danger, and occasionally both time and patience are required for its treatment.

<sup>1</sup> Vide *The Senile Heart*, p. 76, etc.

*Tremor cordis* is a remarkable form of cardiac irregularity, most alarming to the sufferer, yet not apparently attended by any danger. Like a bolt from the blue, and with as little warning, a heart beating quietly and steadily is suddenly seized with a rapid fluttering, and the ordinary full pulse of health suddenly drops to a mere tremulous thread. These attacks vary from three or four short, incomplete systoles rapidly succeeding one another up to a whole series of short, rapid, and incomplete systoles, which may last for several seconds, convey a tremulous sensation to the hand laid upon the cardiac region, and are accompanied by a small fluttering and often scarcely perceptible radial pulse. The attack ends suddenly, like an intermission, with an unusually forcible beat. During all these imperfect systoles the ventricle has been gradually getting overfilled, the augmentor nerve is called into action, and the ventricle forcibly expels its contents, which escape freely and distend arteries which have had time to get unusually empty. This form of irregularity is never attended by any feeling of faintness, and seems always to be associated with the presence of flatulence in the stomach, or with some other gastric disturbance. It never appears to arise from any psychical reflex, and the most singular fact connected with this remarkable symptom is the sudden way in which an apparently healthy heart, beating quite regularly and steadily, begins to flutter in a manner most uncomfortable and alarming to the sufferer. No doubt, in all such cases there must be some weakness of the myocardium, and cardiac tonics are thus indicated, but all direct heart medication must be subordinated to careful dieting and to such treatment as is fitted to improve the digestion and the health generally.

An *unusually slow pulse* is sometimes normal, may be found in very early life, and may continue to the end. The pulse of the great Napoleon, according to Corvisart, was never over 40 per minute; and M. Roux tells us of an agriculturist who had gone through his military service

without difficulty, who never ailed, and was a typical example of good health, yet his pulse-rate was never over 34 to 40 per minute, and even a run of several minutes never raised it higher than from 50 to 55, and that only for a few seconds.<sup>1</sup> Nevertheless an abnormally slow pulse is almost invariably pathological in its character. Hope tells us that "when one or two beats are regularly and permanently imperceptible in the pulse, such cases constitute the bulk of those in which the pulse is described by non-auscultators as being singularly slow—for instance, 30 or 20 per minute." But he adds, "In a few cases, however, it is really slow."<sup>2</sup> Hope is quite correct; there are two varieties of *bradycardia* or slow pulse—one in which the heart is feeble, and one or two of its systoles regularly fail to reach the radial pulse, constituting the "false intermission" of Lænnec, the hemi-systolic bradycardia of modern writers. Cases of true bradycardia belong to quite a different category, and have quite another origin. I have already narrated two cases of hemi-systolic bradycardia, one in which two cardiac systoles regularly failed to reach the radial pulse, so that the heart beat at the rate of 60 and the pulse at 20 per minute (*vide* p. 275). I have also given the sphygmogram of another case in which alternate systoles failed to reach the radial pulse, but in it there was also this other form of irregularity, that the right and left ventricle predominated alternately; the pulse was a *pulsus alternans* (*vide* p. 276). The following sphygmogram is from a third case. In it you will observe that the pulse-wave rises at once to its full height, the secondary dirotic wave occupies its usual position, but lower down the descending limb—just where in a normal tracing a new pulse-wave ought to begin—there is a slight elevation (*a*) due to the imperfect or abortive systole (hemi-systole), imperceptible to touch and not always to be found

<sup>1</sup> *Vide* "Le poulx lent permanent," par le docteur E. Leflaive. *Gazette des Hôpitaux* (1891), p. 1072.

<sup>2</sup> *On Diseases of the Heart*, 3rd edition (London, 1839), p. 377.

in the tracing. In such cases the heart is always feeble and dilated, and sometimes fatty. It was this form of slow pulse, doubtless, that the earlier observers—Adams,<sup>1</sup> Stokes,<sup>2</sup> and Richard Quain<sup>3</sup>—connected sequentially with a fatty myocardium; but the seeming connection is only a coincidence. A fatty myocardium may be accompanied by a slow pulse, but quickening of the pulse, increasing with age, is also frequently associated with the same pathological condition.<sup>4</sup> In these circumstances, as in recovery from febrile conditions, slow and fast pulses are interchangeable, and are indications of cardiac debility, and not of any special degeneration of the myocardium which may, or may not, be present. From the conditions prevalent when this form of slow pulse is present it is evident that the treatment most likely to be useful is a tonic treatment, the special elements being carefully selected with a view to the particular requirements of the heart, with due attention to the needs of the general system. Later on a case will be found narrated in which this treatment was eminently successful.



FIG. 26.

As will be at once seen from the annexed figure, the sphygmogram of true bradycardia is very different from that of the hemi-systolic variety. The tracing (Fig. 27) shows what appears to be a great round-topped predicrotic blood-wave, as if the blood-pressure was greatly increased, or as if a rigid arterial wall was only slowly raised by the advancing

<sup>1</sup> *Dublin Hospital Reports*, vol. iv. 1827.

<sup>2</sup> *Diseases of the Heart and Aorta* (1854), p. 326.

<sup>3</sup> *Medico-Chirurgical Transactions*, vol. xxxiii. p. 162.

<sup>4</sup> *Quain's Dictionary of Medicine* (1894), vol. i. p. 805.



blood-wave. The true explanation is very different from either of these. When the heart beats slowly the ventricles get more distended the longer the diastole is prolonged. At each systole a larger blood-wave than usual is thrown out, and, as the arteries have had a longer time than usual to empty themselves, it passes rapidly onwards; the secondary dicrotic wave is of greater amplitude than usual, and occurs earlier on the descending limb. A glance at the tracing (Fig. 27) shows this very clearly. Here the letter A marks the highest point of the pulse-wave; the rounded top immediately following is not the pulse-wave, as might be supposed, but the secondary or dicrotic wave placed near the summit of the descending limb instead of about its middle. In some bradycardiac sphygmograms this dicrotic wave is so ample and so premature that it seems to occupy the very summit of the pulse-wave, while the true apex of this lies beneath it, giving the tracing an anacrotic appearance.

The passage of the large blood-wave through the arteries is naturally associated with a great rise of blood-pressure, but this is but momentary, as it rapidly dies off from the continuous outflow through the arterioles during the prolonged diastole. Hence in bradycardia, as in aortic regurgitation, we have an exceptionally high blood-pressure alternating with an abnormally low one, a condition that explains much that is anomalous in the history of bradycardia, and has a not unimportant bearing on its treatment.

In the sphygmogram given the pulse-rate is 32, but this patient's pulse varied from 28 to 36; the heart was markedly

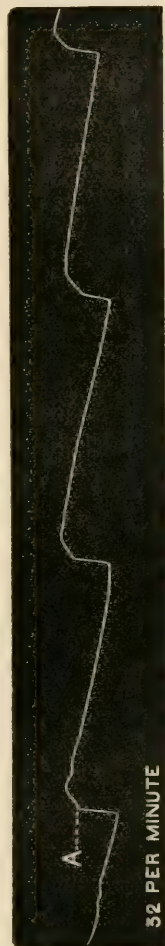


FIG. 27.

dilated, indicated by extension of the præcordial dulness, apex beat to the left of its normal position, and the permanent presence of a systolic mitral murmur, and usually of a systolic murmur in all the cardiac areas. As these slow hearts generally belong to the latter half of life, and, in common with all the other tissues, suffer in nutrition from the extremely low blood-pressure prevailing during the prolonged diastole, and as they have to cope with an extremely high blood-pressure shortly after the commencement of systole, we cannot wonder that they are always dilated. Such hearts are also hypertrophied, but never much; though dilated, they are never weak, rather the reverse.

Roy and Adami tell us that inhibitory impulses through the vagus may slow or even arrest ventricular action for a short time, but that the heart is never permanently slowed in this manner, because sooner or later the ventricles take on a rhythm of their own—an idio-ventricular rhythm, which may be fairly rapid and almost perfectly regular.<sup>1</sup> But though impulses through the vagus are incapable of producing this result, we know that irritation or injury to its important branch, the spinal accessory, may be followed by temporary or permanent slowing of the heart. The spinal accessory rises by several filaments from the side of the cord as low down as the fifth or sixth cervical vertebra, and fractures of the vertebræ in this region are known to be accompanied by slowing of the pulse down to 20 or 30.<sup>2</sup> Concussion of the spine in this region has been followed by temporary slowing of the pulse to between 56 and 48,<sup>3</sup> and Holberton has recorded a most interesting case of injury in this region in which inflammatory compression of the cord occurring after the lapse of a year was followed by permanent slowing of the pulse averaging 33, and sometimes as low as 20, 15,

<sup>1</sup> *Philosophical Transactions*, vol. clxxxiii. p. 293, etc.

<sup>2</sup> Gurlt, *Handbuch der Lehre von den Knochenbrüchen*, 1864; and Jonathan Hutchinson, *London Hospital Reports* (1866), p. 366.

<sup>3</sup> Charcot, *Lectures on Diseases of the Nervous System*, New Sydenham Society edition (1881), p. 117.

or even 8 per minute, and this slow pulse was accompanied by syncopal attacks, ending in epileptiform seizures, in one of which the patient ultimately died.<sup>1</sup> Various disturbances of the chylopoietic functions have been found associated with a slow pulse,<sup>2</sup> and there are many poisons, both organic and inorganic, which slow the pulse, and all of these have a direct action on the nerves and nerve centres. But all the information at present before us seems to point to direct action on the spinal accessory in the neck or within the chest, before or after its junction with the vagus, as an essential requisite for the production of permanent slowness of the pulse.

Such patients generally die in one of their epileptiform attacks, but, though several of my patients have died thus, I have never seen an attack. According to Holberton, a fit is always preceded by cessation of the pulse for a second or two. After this "the face would redden, and consciousness return with a wild stare and occasionally a snorting, a slight foaming at the mouth, and a convulsive action of the muscles of the mouth and face."<sup>3</sup> The initial seizure seems thus to be essentially syncopal in character, and the succeeding phenomena are evidently due to the unusually large blood-wave with which the tissues are suddenly flushed on the return to life. Until more certain information is obtained as to the essential cause of the permanent slowness of the pulse, there seems to be no possibility of any radical cure of the complaint, and the best palliative treatment would seem to be the administration of digitalis in moderate tonic doses, so as to maintain the metabolism of the myocardium, and enable it to withstand the great strain thrown upon it by the conditions under which the circulation is carried on.<sup>4</sup>

*Delirium cordis* is an extreme degree of irregularity apparently always depending upon great dilatation of the

<sup>1</sup> Holberton, *Medico-Chirurgical Transactions* (London, 1841), p. 76.

<sup>2</sup> Burnett, *Medico-Chirurgical Transactions* (London, 1827), p. 202.

<sup>3</sup> Holberton, *loc. cit.* p. 79.

<sup>4</sup> Vide *The Senile Heart*, *sub voce* Bradycardia, also p. 293.

heart, though a very good imitation of it is sometimes found associated with mitral stenosis. It is not very amenable to treatment, and in those cases I have seen relieved this relief has only been obtained by the use of large doses of digitalis continued for some considerable time.

Throbbing pulsation of the ascending aorta accompanies Graves' disease in a marked manner, and is naturally more or less present in all forms of palpitation; but what is generally understood by aortic palpitation is an epigastric pulsation of the abdominal aorta. This is a local neurosis not always apparently dependent on dyspepsia, nor to be relieved by tonics. I have generally found it yield to full doses of the bromides in some bitter infusion. Perhaps the only exception was a woman in Ward XIII., in whom this excessive abdominal pulsation was accompanied by preternatural hardness of that part of the artery, and in her case large doses of iodide of potassium gave great relief, though nothing seemed to have any permanently curative effect.

In hospital you will not probably have many opportunities of seeing any great variety of the many forms of irregularity just described, but in after-life you will fall in with many cases, and it is well to have some information about them. Nothing can make a man feel more helpless than to be brought for the first time face to face with a bad attack of tachycardia, or of *tremor cordis*, of the nature of which he knows nothing, and may perhaps have never even heard of its existence.



## LECTURE XI

### ON SOME OF THE SECONDARY RESULTS OF CARDIAC DISEASE

THERE are many subsidiary affections which—for physiological reasons—naturally group themselves round cardiac lesions, and now and then one or other of these affections asserts itself in so marked and determinate a manner as to challenge attention as if it were a primary and independent disease. It is of importance, however, for the patient, sometimes for his safety and always for his comfort, that we should recognise these ailments as secondary, and by at once attacking the primary cause give speedily that relief which, if obtained at all, can only be obtained in a round-about and unsatisfactory manner if we treat as a disease what is essentially a symptom.

The ailments to which I refer are inseparably connected with those alterations in the circulation which begin with the very commencement of cardiac disease, and upon the greater or less rapidity with which they advance its termination depends. These circulatory changes may be grouped under two heads—*first*, changes in the intra-arterial blood-pressure; and, *second*, venous remora.

The absence of capillary resistance and of vascular motor nerves within the lungs make mere mechanical influences play a more important part in the pulmonary than in the systemic circulation. Hence, as we know, any obstruction to the onward flow of the blood through either lungs or heart is speedily revealed by an accentuation of the second sound at the orifice of the pulmonary artery,<sup>1</sup> and this is speedily

<sup>1</sup> *Vide* Lecture I. p. 30.

followed by dilatation and congestion of all the pulmonary vessels thus predisposing to catarrhal affections of the bronchial mucous membrane. In time the pulmonary capillaries become tortuous and varicose, encroaching on the air-cells, limiting the air space, and, coupled with extravasations into the parenchyma, forming what is known as brown induration of the lung. Hence in all heart affections of long standing much of the breathlessness complained of is due to this condition of the capillaries, and this may be greatly aggravated by many causes, such as catarrh of the bronchial membrane, an oedematous condition of the air-cells themselves, or by serous effusion into the pleura or pericardium, or even by the great size of the heart itself, not to mention the impoverished (spanæmic) condition of the blood due to the long continuance of defective hæmogenesis.

In all cardiac cases there is a generally imperfect metabolism; this induces a defective vitality in the walls of the vessels, especially at certain parts, as in the neighbourhood of the venous valves, and this, coupled with the general remora of the venous circulation and its diminished intensity everywhere, often gives occasion to spontaneous coagulation of the blood in dilated bulgings of the systemic veins, in the arteries also, especially if atheromatous, and even in the trabeculæ of the ventricles and also of the auricular appendices. Portions of these *thrombi* often get detached and carried as *emboli* into other parts of the circulation. Sometimes they block an artery, causing gangrene of the part to which the vessel is distributed; more often arterial blocking is caused by the spreading of an autochthonous thrombus. Coming from the heart or veins such emboli are sometimes large enough to block the pulmonary artery and cause sudden death, or a less complete occlusion of one or more branches of that vessel may lead to a more gradual manner of dying. If an embolus drops into a lung—and the lower part of the right lung is from circumstances the most common position, though an embolus may be found in any part of either lung—it gives

rise to an infarction, a local stasis often associated with a sudden but temporary rise of temperature, a small localised patch of dulness, over which crepitation is to be heard; occasionally there is slight pain in the chest, and sometimes hæmoptysis. An infarction of a different character may be produced by rupture of a vessel, often of some size, within the lung; in this case the blood is poured with a force corresponding to the amount of intravascular tension present into the bronchus with which the ruptured vessel is connected, filling every part of the corresponding lobule as far as its terminal sacculi. It may happen indeed that more than one lobule is thus filled before coagulation finally takes place, so that an infarction thus formed is often much larger than one resulting from an embolus. We recognise this by the larger size of the dull area lying over the part affected, and of this we are also forewarned by the very considerable hæmoptysis that accompanies this form of pulmonary apoplexy if it be at all important.

The infarction that follows the impaction of a perfectly healthy embolus gives rise to a wedge-shaped induration, varying in size, colour, and consistence, according to its age. Sometimes, however, an embolus may come from a septic thrombus, or a purely hæmorrhagic infarct may become infected by septic germs, and the result in either case may be the production of a localised (embolic) pneumonia, which may lead to suppuration or gangrene; is always serious and often fatal, and accompanied by all the usual symptoms of gangrene of the lung, and often by those of pneumothorax or hydro-pneumothorax as well.<sup>1</sup> The consciousness that such serious ailments may result from cardiac disease does not, unfortunately, enable us to prevent them, but it ought to be an incentive to us not to limit our attentions to the care of our patient's myocardium, but in every way to endeavour to maintain his general health in as perfect a state as possible.

<sup>1</sup> *Vide* Cohnheim's *Pathology*, New Sydney Society edition, chapter iv. p. 172, etc.

From the frequency with which hæmorrhagic infarctions occur in the course of cardiac disease hæmoptysis is quite as often dependent on cardiac as on pulmonary disease, and even when actual disease of the lung is present the co-existence of cardiac disease, especially if that be a mitral stenosis, exercises a most important influence upon the hæmorrhage. In cases of frothy serous expectoration with rhonchi and crepitation in the lungs, we have to look to cardiac debility—if not to some more serious lesion of the heart—as only too frequently the cause of that pulmonary congestion that has so dangerously intensified the result of some trifling chill. True cardiac breathlessness presents symptoms so altogether peculiar that it can scarcely fail to be recognised and its central cause detected.<sup>1</sup>

It is, however, in the systemic circulation that we find the most multifarious phenomena dependent on the one central lesion—phenomena which are as varied as the functions of every organ in the body, lesion of any one of which is their proximate cause. Confining our attention for the moment to the results of defective blood-pressure, I need scarcely insist upon the importance of due recognition of the cause of those pseudo-apoplectic or epileptiform seizures I formerly discribed<sup>2</sup> as the result of failure of the heart's force, or of the giddiness, threatened or actual syncope, *tinnitus aurium*, flashings of light across the field of vision, most noticeable by the patient himself on stepping into darkness from light, and occasional flushings of a usually pale face, which the patient describes as “puffs of heat,” which are all indications of cardiac debility of common occurrence and in their simplest forms due to anæmia, in their most severe forms to aortic regurgitation, while simple cardiac dilatation is a common cause of intermediate varieties. When accompanying aortic regurgitation these symptoms are combined with a full bounding pulse and are in danger of being

<sup>1</sup> Lecture I. p. 2.

<sup>2</sup> Lecture X. p. 261.



referred to cerebral congestion of an acute character, and are liable to be treated rather differently from what they require as mere indications of cardiac debility. Having already gone into the treatment of such cases<sup>1</sup> it is unnecessary to recapitulate this at present; I have merely referred to them as examples of the many and various symptoms which, though not apparently implicating the heart, are yet important indications bearing not only on the need for a careful examination of that organ, but also on the necessity of guiding our treatment by its condition, because it is only by rectifying the central lesion, so far as that is possible, that we can expect to remedy the peripheral phenomena depending upon it.

Failure of secretion is, as we all know, a common indication of failure of blood-pressure in the dying, but we ought to remember that diminished secretion is also a usual accompaniment of that lowering of the intra-arterial blood-pressure that is inseparable from cardiac disease. Perhaps the earliest of the secretions that fail from this cause, certainly the one that is earliest noticed to fail, is the secretion of the kidneys. Unfortunately this may be well enough known to the patient but not to his physician; the patient himself is unaware of the importance of such an apparently trifling fact and omits to mention it, but it ought always to be inquired into as an early indication of cardiac failure, often of extreme significance, especially in heart cases of long standing which may present no other indication of loss of power. Whenever we find the urine persistently diminished in quantity, and its specific gravity unaltered or even increased, and when we find as the result of the diminished quantity of water a deposit of lithates, apart from any coldness of the weather or of any somatic catarrhal or febrile condition, then the condition of the central organ should be inquired into; it will certainly be found defective. When the water of the urine is diminished a certain soakage of the tissues takes place *pari passu*, but

<sup>1</sup> Lecture III. p. 102.

this is so uniformly distributed throughout the tissues that it is unobserved, and is probably only revealed by increased breathlessness otherwise unaccountable. The salivary glands are the only other organs in which any deficiency of secretion would be equally readily detected. But, as is well known, they are, so far as secretion is concerned, entirely separated from the general circulation and placed under local influences, so that any deficiency in their secretion is wholly deprived of any somatic importance, so far at least as the heart is concerned.

The gastric secretions are not so readily measured as those just referred to, but the results of their deficiency are very evident, especially to the patient himself. The flatulent stomach due to gastric secretions deficient both in quantity and quality has often been unavailingly treated by tonics, blue pills, antispasmodics, etc., the central lesion—even when recognised—having been ignored in the treatment, not having been regarded as the efficient cause of this subordinate phenomenon. Yet in all such cases our first inquiry ought to be into the state of the urine, as a corroborative indication, and our next investigation into the condition of the heart itself. It is astonishing how long such subsidiary phenomena may persist without progressing farther, provided there is no important ailment beyond myocardiac weakness. I have seen a flatulent abdomen and defective secretion of urine last for twenty years without the development of any more serious symptom, if we except considerable marasmus, which was to be expected from the long-continued imperfect digestion. And I have also seen all the more prominent symptoms in a similar case vanish after a few days' appropriate treatment.

If we except the cerebral phenomena which may accompany anæmia or may be symptomatic of aortic incompetence, it is seldom that we find symptoms due to defective blood-pressure apart from others associated with venous congestion, and sometimes the latter alone predominate. Thus in females we frequently find menorrhagia due to the venous congestion

arising from cardiac disease, and in both sexes we may have hæmorrhoids due to congestion of the portal system, the result of the remora in the systemic venous circulation. Whenever there is, from any cause, failure of cardiac energy, not merely in the later stages of cardiac disease, the same venous congestion of the kidneys that in a lesser degree causes diminution of the urine in a greater degree causes the urine to be contaminated with serum-albumin, to a greater or less extent according to the degree of congestion present. The greater or less amount of serum-albumin found in the urine of such cases depends upon the increased intra-venous pressure. From the researches of Kürschner<sup>1</sup> we know that fluids pass through animal membranes in the following order: *first*, water; *second*, saline solutions; and, *lastly*, albuminous or gummy fluids. Bruecke<sup>2</sup> has also shown that certain membranes allow albuminous fluids to pass more readily than others, and that this probably depends upon the size of the pores. And Liebig<sup>3</sup> has confirmed Kürschner's experiments by measuring the force necessary to drive the various fluids through membranes. In the course of this inquiry he ascertained that the pores became apparently widened by a continuance of the experiment, so that after the lapse of twenty-four or thirty-six hours the force of the pressure might be reduced by one-third or even by one-half, without any diminution of the result. Widening of the pores of any membrane must thus be regarded as the proximate cause of the passage of albumin through it, and we cannot but consider the intra-venous pressure of long-continued remora due to cardiac debility as a most efficient cause of albuminous transudation.<sup>4</sup> Moreover, as long per-

<sup>1</sup> Wagner's *Handwörterbuch der Physiologie*, Bd. i. S. 62.

<sup>2</sup> *De diffusione Humororum per Septa Mortua et Viva* (Berol. 1842), S. 55.

<sup>3</sup> *Untersuchungen über einige Ursachen der Säftebewegung* (Braunschweig, 1848), S. 6; *vide* also Henlé's *Rationnelle Pathologie* (1847), Bd. ii. S. 467-8.

<sup>4</sup> Robinson, *An Inquiry into the Nature and Pathology of Granular Disease of the Kidney* (London, 1842); Emmert, *Beiträge*, etc., and Henlé's *Rationnelle Pathologie*, Bd. ii. S. 458, etc.

sistence of pressure is, as we see, equivalent to considerable increase in amount, and as we learn from Kürschner's experiments that membranes permitting the passage of saline fluids are but one step removed from those permitting the passage of albuminous fluids, we can readily understand that the transudation of serum-albumin from the kidney is easily brought about by any increase of intra-venous blood-pressure so liable to occur from various causes in the course of cardiac disease. We must, however, remember that diminished secretion is the most sensitive test of defective cardiac energy, that this depends upon deficient intra-arterial blood-pressure, and that to render increased intra-venous pressure effective in promoting albuminous transudation a certain degree of vascular fulness is requisite. Hence if great marasmus is present, diminished secretion may persist for many years without the occurrence of albuminous transudation; while if much vascular turgescence be present, albuminous transudation may be a comparatively early symptom. Even a small amount of albumin in the urine is therefore always an important sign of cardiac failure, when observed apart from any other efficient cause. Hyaline tube casts have no other significance, as a rule, but the presence of epithelial casts may be looked upon as an indication of the co-existence of independent disease of the kidney. Besides, a diminished amount of urine of a normal or high specific gravity, with a small amount of albumin, is not found in any independent form of kidney disease of any extent. A diminished amount of urine with a large amount of albumin indicates inflammatory disease of the kidneys, which has also other and more distinctive symptoms. A varying, but usually large amount of albumin with a large quantity of nearly normal urine, indicates waxy degeneration of the kidneys, while in contracting kidneys, which have a very close and intimate connection with cardiac and especially with arterial disease, the urine is never more than slightly albuminous, and its quantity tends to increase and the specific gravity to be low, though



in the early stages there may be but little change in either respect. The albuminuria of cardiac congestion thus differs entirely from that of true renal disease, even in the contracting form in which there seems to be generally some degree of cardiac failure, the history of the case is such as to preclude any possibility of a mistake.<sup>1</sup>

Whenever there is imperfect digestion from a gastric juice deficient in quantity and defective in quality due to cardiac debility, there is also venous congestion of the liver, a diminished secretion of bile, congestion of the portal circulation, and a tendency to hæmorrhoids, diarrhœa, or ascites. The deficiency of bile intensifies the results of the imperfection of the gastric juice, hence we have putrefaction of the ingesta, abdominal flatulence, and constipated bowels, with foetid stools. German authors have assumed that at this stage the bile is not only diminished in quantity, but is also diluted with serum (albumincholie), and thus reduced in quality. This is very unlikely to be the case in the early stages of cardiac disease, though it doubtless happens later on when signs of serous escape (œdema) are found elsewhere. In cardiac disease this can only rarely happen first at the liver.

As in the congested lungs catarrhal conditions are apt to be superadded, so also in the congested intestines we are liable to have similar results. But as the bowels are better protected from the vicissitudes of temperature, they are comparatively seldom affected in the early stages of cardiac disease, though in the later stages we occasionally have an obstinate sero-mucous diarrhœa which patients are prone to look upon as an independent disease and to dignify with the name of dysentery. The extension of this catarrhal condition into the bile ducts is apt to produce more or less complete

<sup>1</sup> The connection between the gouty kidney and the gouty heart seems to require serious revision; vide *The Senile Heart*, p. 196. And in regard to this there appears not a little to be learned from the history of the so-called *albuminuria adolescentium*, which at least occasionally passes through the stage of venous engorgement into the true contracting kidney.

obstruction, and so to cause jaundice—a jaundice readily distinguished as of cardiac origin by its greenish hue, the cyanotic tint of the congested skin combining with the bright yellow of the bile pigment to produce this peculiar colour, and perhaps there are few things more striking than the change from green to bright yellow which takes place on the upper surface of the body after death in such cases, from *post-mortem* hypostasis (sugillation).

Whenever there is long-continued congestion of any organ, there must naturally be persistent turgescence of that organ. From this cause alone there is, in some cases, considerable temporary enlargement of the part affected, where that is possible,<sup>1</sup> and ultimately we have, as the result of the venous congestion, hyperplasia of the connective tissue. This overgrowth of the connective tissue is observed to a variable extent in the lungs, more frequently and to a greater extent in the liver, to a less extent in the spleen, and also in the kidneys. Sir William Jenner<sup>2</sup> has taught us that this hyperplasia of the connective tissue is the direct pathological result of long-continued mechanical congestion in any organ, also that this new-formed tissue ultimately contracts, and by destroying the secreting tissue of the organ induces atrophy of the gland affected. But this is by no means a common result of congestion in ordinary valvular lesions; either the disease is fatal before this stage has made much progress, or, as in the case of contracting kidneys, some co-existent ailment—as gouty thrombosis—is present to hasten the climax.

Every case of cardiac disease involves several of these

<sup>1</sup> As in the liver, in which such congestive enlargements connected with cardiac disease often appear and disappear in an incredibly short space of time. *Vide* “*Volumsschwankung des Herzens mit Schwankung des Pulses; korrespondirende Volumsschwankung der Leber und der Milz*,” von Dr. M. Heitler, *Wiener medizinischen Wochenschrift*, No. 13, 1896.

<sup>2</sup> *Vide Medico-Chirurgical Transactions*, vol. xliii. p. 199; and Dickinson's *Diseases of the Kidney*, etc., part ii. p. 385, etc; also Schmaus und Horn, *Ueber den Ausgang der cyanotischen Induration der Nieren in granular Atrophie*, Wiesbaden, 1893; also *The Senile Heart*, p. 196, etc.

secondary lesions to which I have just referred; in different cases we find some of these less and others more marked than usual. In the following case several of these subsidiary affections were conjoined in a well-marked manner.

CASE XXVIII. Elizabeth M'Dermot, aged forty, admitted to Ward XIII. on 30th December 1869, complaining of pain in the chest and breathlessness. She stated that since the February previous she had been subject to pain first in her right hypochondriac region, and latterly over her heart, and that this pain had been more severe during the last four weeks. She never had rheumatism nor scarlatina, but for the last five years she had dysenteric attacks every now and again. She had been in India, but only for a short time, and she was always best when at sea. Patient was of ordinary height (a little over five feet), anxious expression, lips, gums, and conjunctivæ blanched, and tinged of a light yellow; the whole surface of the body was of a waxen yellow hue, the extreme anæmia accounting for the entire absence of the usual greenish colour; her pulse was 98, small and feeble. On placing a hand over the præcordia a distinct heaving impulse was communicated to it by the heart's action, and a thrill was felt immediately preceding and running up to the apex beat. The heart's apex was felt to beat between the fifth and sixth ribs about three inches to the left of the sternum. One inch to the left of the sternum vertical dulness was found to commence at the upper border of the third rib and ran down to the liver dulness. Transverse dulness at the level of the fourth rib commenced at the right edge of the sternum and extended to the left for a distance of four inches and a half. Over the apex a loud, rough, purring murmur was heard preceding and running up to the first sound. In the aortic area both sounds were distinct, the second somewhat feeble. In the pulmonary area the first sound was distinct; the second markedly accentuated. The patient had great difficulty of breathing, complaining especially of oppression on the right side; she had consider-

able cough, and expectorated a quantity of frothy serous fluid. The pulmonary percussion was normal, and on auscultation rhonchi and rattles were heard over both sides of the chest. The tongue was pale but tolerably clean; the patient had no appetite, but considerable thirst. The bowels were irregular, with frequent attacks of watery diarrhœa; the stools were always pale (clay-coloured). The liver dulness extended in the nipple line from the upper border of the fourth rib to about one inch above the umbilicus, a distance of over five inches. The uterus was healthy. The urine small in quantity, deposited urates, and contained a trace of albumin. The prognosis from the first was unfavourable,<sup>1</sup> chiefly on account of the anæmic and exhausted condition of the patient, and she died upon 8th January 1870, her death having been hastened by a recurrence of the diarrhœa. At the *post-mortem* examination the body was not much emaciated; the skin was of a yellow tint. An unusually large quantity of yellow serum was found in the pericardium. The surface of the heart was fatty; the pulmonary valves were competent; the segments of the tricuspid valve were slightly thickened at their free edges; the segments of the mitral valve were thickened, and the auriculo-ventricular opening was narrowed; the segments of the aortic valve were thickened, but the valve itself was competent; the substance of the heart was pale, and apparently fatty. The bronchi were filled with frothy yellow mucus, particularly on the left side; both lungs, but especially the right one, had pigmentary deposits scattered through them, and the parenchyma was stained a yellowish hue; they were otherwise healthy. The liver weighed 4 lbs. 5 oz.; its tissue was slightly fatty and stained yellow. The gall bladder was enlarged and full of dark bile; the bile ducts were œdematous and blocked with

<sup>1</sup> As a rule jaundice is a late phenomenon in cardiac disease, and indicates a speedy end. But I have known concomitant jaundice and dropsy to be both recovered from even when associated with mitral stenosis, and I have also observed jaundice to persist for several years even when concomitant with an ultimately fatal stenosis of the mitral opening.



yellowish mucus. There were many embolic cicatrices on the surface of the right kidney; the renal tissue was paler than usual and of a yellowish tint; it was slightly fatty, and the Malpighian corpuscles somewhat prominent. The spleen weighed  $8\frac{1}{2}$  oz. and had a lobulated surface; on being cut into its tissue was firmer than usual, but there was no alteration beyond simple hypertrophy.

The general venous turgescence is not, however, only to be recognised by alterations in the secretions, or in the ultimate changes of structure that result from it. It is readily discerned in the bluish or purplish tint of the lips, cheeks, and digital extremities, and in rarer cases by a dusky hue—cyanosis—that overspreads the whole surface of the body. Now and then, when the superficial congestion is not extreme, and the skin is delicate as in young females, a certain amount of oxygenation seems to take place through the more exposed parts, and the cheeks may have all the rosy hue of health, yet even in these cases the digital extremities are always blue and cold; indeed the surface temperature of the body is usually somewhat depressed. Occasionally some of the larger external veins become turgid, but congestion must be considerable before this takes place, and it is not very common in young persons in whom the external veins are generally small. In the neck, as I have already told you, the veins not infrequently pulsate, and pulsation may now and then—though rarely—be seen in all the superficial veins.

In all cases of cardiac disease the tissues are more or less soaked with serum, and very trifling causes may precipitate the occurrence of pulmonary œdema or pleural effusion. The latter condition is revealed by a rise in the percussion note, and a diminution of the pulmonary murmur. A slight alteration in the force or distinctness of the pulmonary murmur on one side as compared with the other, without change in the percussion note, indicates pulmonary œdema long before any crepitations are to be heard. These changes

are more common in connection with mitral than with aortic disease, as in the latter they rarely occur until the mitral valve also has become implicated. Sooner or later in most cases anasarca sets in, commencing at the most depending part, usually the instep, and gradually increasing till it accumulates more or less in every cavity of the body, constituting what is called general dropsy. The instep may thus be called the position of election for the commencement of cardiac dropsy, yet when the patient from any cause is bedridden, œdema is first found over the back, or it may be in the pleuræ, or if cardiac congestion has lasted for long without any serious cardiac debility, hepatic obstruction or even some degree of cirrhosis may be sufficient to determine the earliest indication of dropsy to be within the peritoneum instead of the connective tissue, so that we have ascites instead of anasarca as the commencement of true cardiac dropsy. Apart from the independent yet concurrent occurrence of hepatic disease, this condition is limited to mitral disease, and chiefly to mitral stenosis.

Embolism is common enough in the course of cardiac disease, and gives rise to phenomena of the most diverse character, according to the organ affected. Thus we may have paralysis of motion, sometimes temporary, frequently permanent; paralysis of sensation alone, rarely; aphasia, often; hæmaturia, followed by temporary albuminuria, not infrequently; hæmoptysis, with pulmonary infarction, very frequently; not seldom complicated by pneumonia or effusion into the pleural cavity, besides a host of pains and other anomalous symptoms that frequently depend upon embolism, though the only obvious connection may be the presence of cardiac disease and the suddenness of the attack.

Embolism, as you know, is the term applied to a local stoppage of the circulation by the sudden blocking of a vessel by an embolus, that is by some foreign body carried along in the blood-torrent. A few air bubbles or oil globules may, under certain circumstances and in certain situations, produce

all the obstructive effect of more solid masses. More usually, however, the obstructing mass is a bit of bloodclot, or a fibrinous concretion from a vein, from an aneurysm, or from the cavity of the heart, or a vegetation from a diseased valve. The phenomena of embolism vary in each particular case; they depend upon the size of the embolus, as well as on the function of the organ in which it happens to be arrested. Yet varied as the results may be, there are probably few pathological problems more simple in their essence, or the steps of which can be so readily followed.

Perhaps the most instructive way in which, not to be tedious, I could conclude this lecture, will be to narrate the following case, in which repeated embolisms of various organs occurred during the patient's residence in hospital. The case itself is otherwise of considerable interest, and in spite of the absence of a *post-mortem* examination, there can be no reasonable doubt as to the cause of the repeated attacks.

CASE XXIX. J. M., aged thirty-three, a wardress in the Edinburgh Prison, was admitted to Bed 16, Ward XIII., on 17th April 1873, complaining of pain in her left side, extending down her thigh to her calf, and upwards to her left forearm and hand. These pains had troubled her continuously for three months; they had been accompanied by repeated diurnal rigors and nocturnal perspirations, and had been treated as rheumatic by her medical adviser. The patient's family history was good, and she herself had up to the present time enjoyed good health, except that she had suffered from a severe attack of rheumatic fever at the age of fifteen. Her expression was weary and anxious; she was nervous and easily startled, had occasional headaches, and slight hæmorrhages from her nose when blown. Her respirations were 32 per minute; pulmonary physical signs normal; no cough nor expectoration. Her pulse was 96, soft and easily compressible; its systolic impulse was forcible, and it fell rapidly off from the finger (Corrigan's pulse)—a phenomenon markedly increased by elevating her arm at a right

angle to her body as she lay in bed. The heart's apex beat full, diffuse, and forcible, below the sixth rib, two and a half inches from the left edge of the sternum. At the level of the fourth rib dulness commenced one inch to the right of the sternum, and extended across the chest for a distance of five and a half inches. The cardiac vertical dulness did not rise above the third rib, but the aortic dulness extended to the upper edge of the sternum, and the aortic pulsation was distinctly to be felt in the tracheal fossa. On listening in the mitral area a rough murmur was heard running up to the first sound, which was obscured, but not replaced, by a systolic murmur which, when traced upwards, was found to have its position of maximum intensity in the aortic area. Here this murmur was loud, rough, and followed by a soft, blowing diastolic murmur, which wholly replaced the second sound. In the pulmonary area there was a distinctly accentuated second sound somewhat obscured by a diastolic murmur propagated across from the aortic area, and preceded by a systolic murmur coming from the same region. In both carotids a distinct double murmur was to be heard. The patient's tongue was clean, appetite moderate, bowels regular. The urine was slightly opalescent, and of a pale straw colour, moderately acid, without deposit, but containing a trace of albumin; its specific gravity was 1026. The patient had not menstruated for several months. Here there could be no dispute as to the diagnosis; evidently the mitral opening was constricted from disease of the valve, and the aorta dilated with a diseased and incompetent valve, but from the signs present its segments were not probably atheromatous to any extent, but only crumpled or shrivelled. Both valve troubles were evidently due to her rheumatic attack eighteen years previously, her present illness being possibly but doubtfully of rheumatic character. The prognosis was moderately hopeful as to the issue of her present illness, but serious as to the ultimate result of her cardiac disease.



The double murmur in this case was readily audible in the carotid arteries, indicating great freedom of regurgitation, and therefore absence of any marked thickening of the aortic segments, and as the affection was evidently of rheumatic origin, and unaccompanied by any great dilatation of the aorta itself, it was obvious that the valve segments must be shrivelled or retracted to permit of regurgitation so free as that which existed. The double murmur in the aortic area, with its first element loud and rough, indicated local obstruction to the egress of the blood as well as to its ingress; pointed therefore to deformity of the valve segments with some stiffening of their structure, and, as so often happens in similar cases, probably some vegetations on their under surface. Were the valve segments comparatively normal with so free regurgitation as we had in this case, the signs would have been entirely different. In that case the double murmur would have been audible in the arteries alone, with a single diastolic murmur to be heard below the level of the valve, and a comparatively mute aortic area. The persistent localisation of the neuralgic pains was opposed to any idea of them being purely rheumatic in character, while the alternating rigors and sweatings were evidently more neurotic than rheumatic in character, and the condition of the urine agreed with this supposition. As the case subsequently developed there seemed reason to suppose that the illness was most probably entirely embolic in character. The patient continued to improve till 17th May, exactly one month after admission, when she had a sudden rigor. She was much distressed, and complained of intense headache. Her temperature rose suddenly to  $105^{\circ}$ ; her pulse to 112; and her respirations to 40. The urine contained a trace of albumin; the chlorides were apparently undiminished. On examination of the chest a small localised patch was discovered at the base of the right lung posteriorly, over which fine crepitation was heard at the end of inspiration. No dulness could be found on percussion; there was no bronchial

breathing; no cough and no expectoration. A jacket poultice was applied to the chest, and ten minims of chloroform in one drachm of olive oil were given every four hours.

18th May.—Temperature, *mane* 104·6°, *vespere* 103·6°; respirations 40; pulse 108. Distinct crepitation was audible on inspiration over the base of the right lung; no dulness; no bronchial breathing, nor bronchophony; slight cough; no expectoration.

19th May.—Temperature, *mane* 102°, *vespere* 101·8°; respirations, *mane* 36, *vespere* 28; pulse, *mane* 96, *vespere* 88; crepitation on inspiration still present; urine slightly albuminous; chlorides undiminished; tongue foul.

20th May.—Temperature 98·8°; respirations 32; pulse 84; crepitation no longer to be heard; pulmonary physical signs altogether normal.

The suddenness of this attack, and its equally rapid recession, correspond to the peaked ephemeral type of pneumonia, a type occurring, as Wunderlich has pointed out, chiefly in connection with trifling local processes, and frequently of embolic origin. In this case the embolic origin of this sudden and sharp attack was at once recognised and pointed out, no credit being taken for cutting short a disease the pathological course of which is generally so brief. In the present day one scarcely requires to point out how similar cases in olden times may have served to bolster up the idea that the pneumonic process might be suddenly aborted by active perturbative treatment. Had our patient perchance been freely bled, with what chucklings might we not have recorded the obvious recurrence of the good old type in her pneumonia, and its remarkable amenableness to appropriate treatment.

The patient remained well till the evening of 1st June, when she was suddenly seized with sickness and vomiting. Her tongue was foul; headache intense. Nothing abnormal could be found anywhere except in the urine, which was smoky, with a red deposit of blood corpuscles, as recognised

by the aid of a microscope; no tube casts were observed this evening, but the examination was somewhat hurried. The urine contained about one-seventh of albumin; chlorides undiminished. A mustard poultice was applied over the epigastrium, and small bits of ice were directed to be swallowed occasionally.

2nd June.—Patient in much the same state; vomiting continues, and headache still intense; urine bloody, specific gravity 1015, albumin about one-seventh; epithelial and granular casts were to-day observed for the first time, and in considerable number; pulse, *mane* 92, *vespere* 104; respirations 37; temperature, *mane* 100·2°, *vespere* 101·6°.

*Treatment:* Potassii iodidi,  
Ammonii bromidi, aa ʒ ii.  
Infusi chimaphilæ, ʒ vi. Solve.

Signetur—Capiat æger sem'unciam tertiis horis ex aqua.

3rd June.—Headache still severe; vomiting ceased; a vesicular eruption beginning to appear on the forehead and cheeks, urine unchanged; pulse, *mane* 114, *vespere* 120; respirations ranged from 32 to 36; temperature, *mane* 102°, *vespere* 103·4°. Omit the iodide.

4th June.—Eruption well marked, forming transparent bullæ ranging in size from a split pea to that of half a (boy's) marble, and full of limpid fluid (Hydroa); headache lessened; urine unchanged, tube casts more numerous, and more of them granular; tongue dry; pulse 108; respirations 36; temperature, *mane* 105°, *vespere* 104·2°.

5th June.—Headache gone; urine unchanged; patient complains of no pain, but is evidently intensely ill; pulse, *mane* 116, *vespere* 114; temperature, *mane* 102°, *vespere* 102·4°; respirations, *mane* 41, *vespere* 34.

After this the patient went on improving till 13th June, when her pulse was 86, her temperature 99·4°, and her respirations 25; tongue clean and moist; appetite good; the blood has disappeared from the urine, which now contains a mere trace of albumin, an occasional granular tube

cast being still to be detected. The eruption, obviously due to the iodide of potassium, has now almost disappeared. This sudden attack of hæmaturia, unaccompanied by any other indications of renal congestion, was evidently due to renal infarction following the arrest of an embolus in the kidney. After the failure of the iodide of potassium the treatment was purely expectant, and consisted chiefly in the free administration of diluents.

On 16th June her temperature was  $98.4^{\circ}$ ; respirations 24; pulse 92. At the evening visit that day, while the patient herself was under examination, her right hand and arm were seen to twitch convulsively, and the patient, who had just previously been interpreting the language of an aphasic neighbour, became suddenly sick, and complained of violent headache, chiefly on the right side.

Next day, 20th June, she felt better, but still complained of headache, and could not lift her head from the pillow without becoming sick. Temperature  $99^{\circ}$ ; pulse 100; urine free from blood, and containing only a trace of albumin; specific gravity 1020. Nothing else abnormal was observed.

21st June.—Pulse 100; temperature  $100.8^{\circ}$ ; tongue very foul; urine darker than usual, with a copious deposit of phosphates, and almost free from albumin. The abducens muscle of the left eye is to-day noticed to be sluggish in its movements, so that a slight squint is occasioned.

The patient continued to improve till 28th June, when the squint had almost entirely disappeared, the abducens muscle having almost completely regained its normal power. This sudden attack of local paralysis could obviously be ascribed to nothing else than the arrest of a small embolus in the brain, which rapidly broke up and was removed without producing anything more serious than a temporary paralysis of a single muscle; the convulsive movements of the right arm were, like the headache, probably due to reflex irritation.

On 3rd July, at 11.20 A.M., this patient was suddenly



seized with violent headache, chiefly referred to the occipital region, followed by severe vomiting; her eyes were fixed at first, but this passed off, and the patient retained her consciousness for a time. At last, after a violent fit of retching, complete unconsciousness set in, followed by gasping inspirations, which gradually became less and less frequent, the face getting more and more livid, till death occurred at 1.30 P.M. During this final attack the right carotid was seen to beat with much greater force than the left one. Obviously there had been at first a small cerebral embolus followed by a much larger one on the right side, by which so much blood was cut off from the brain as to produce immediate unconsciousness, speedily followed by death from asphyxia from anæmia of the respiratory centre in the medulla. There was no syncope; the heart beat forcibly to the last. No examination of the body was permitted, but the symptoms of each attack were so evidently embolic in character, and the cardiac lesion supplied so efficient an explanation of these emboli, that the pathological history of this most interesting case could scarcely have been made plainer by a dissection.

## LECTURE XII

### UPON PAROXYSMAL ANGINA PECTORIS AND OTHER FORMS OF CARDIAC PAIN, WITH SOME REMARKS UPON THE DIAGNOSIS OF FATTY HEART

PAIN in the cardiac region is a common enough symptom in all forms of diseases of the heart, and is specially apt to be associated with particular varieties; it is often enough complained of when no cardiac disease is present; and it is an important indication of substernal aneurysm. Pain in the region of the heart is always alarming; it reminds one so unpleasantly of angina pectoris, of which John Hunter died, after years of suffering no doubt, but which also cut short the life of Arnold of Rugby in a first attack. All attacks of angina are not equally serious, but all have within them the elements of danger; fortunately every pain in the region of the heart is not angina. Hence some have classified all cardiac pains under two heads—angina and pseudo-angina—the latter term being apparently employed to hedge the diagnosis, and give it a semblance of truth however the case may turn out.

For my own part I recognise a set of symptoms indicative of angina pectoris, of which the most alarming is not pain but an overwhelming sense of impending dissolution. But angina pectoris is not a disease: it is merely a syndrome; and the disease that underlies it is sometimes revealed by a very different syndrome, of which the most striking characteristic is seemingly causeless breathlessness—cardiac asthma; while in yet another set of cases the disease is

suddenly fatal, unpreceded by any recognisable symptoms—*angina sine dolore*. And I also recognise another class of cases in which pain is the predominant symptom, and in which any anxiety present is due to the fact that the pain lies in the region of the heart. Sometimes this pain can be referred to a definite cause and labelled with a distinctive epithet. At other times no very evident cause is detectable, and these cases I prefer to leave unlabelled for a time. My experience is that after a longer or shorter period the cause is sure to be revealed, and it seems better to wait and watch rather than stifle inquiry by giving to the pain an epithet so apparently satisfactory and yet so meaningless in reality as *pseudo-angina*.

When a patient comes to us complaining of pain in the region of the heart, our first step is to ask him to point out exactly where he feels this pain, and whether it is distinctly localised or radiates in any particular direction. Next we inquire whether the pain comes on while the patient is at rest or only after exertion; whether it is felt only during the day or at night also; and whether it is most apt to occur after a full meal or has no special relation to the state of the stomach. We also inquire as to the patient's age and contrast this with his general appearance, and further note whether he complains of any other cardiac symptoms, such as breathlessness, etc.

Just recently an elderly gentleman stepped into my consulting room complaining of a sharp pain in the region of the heart, which came on at first during the night, but recurred during the day, and indeed was, when I saw him, never much away. Neither food nor exertion seemed to influence it in any way. The patient did not complain of breathlessness or of any other cardiac symptom; his pulse was small, feeble, and rather quick, but regular. On examination the heart was found to be normal in size, with a feeble impulse, and the sounds weak but pure. There was no oedema of the ankles or lungs, no cough nor any difficulty

or pain connected with the respiration or with any movements of the chest or spine. The pain was thus *per viam exclusionis* resolved into an intercostal neuralgia, and on tracing the fifth intercostal nerve upwards and backwards, a patch of Herpes Zoster (shingles) was found on the lower angle of the left scapula, not very large, but quite sufficient to account for the pain. The patient left me much reassured; his pain was relieved by appropriate treatment, and in a few weeks he was quite restored to health.

Now suppose this patient had no shingles, we should have had to make our diagnosis of intercostal neuralgia all the same, and to connect it—apart from disease of the spine, improbable at his time of life—with some constitutional poison, such as malaria, gout, or syphilis. In malaria we should look for a history of some antecedent exposure to some source of malaria, and there would probably also be some periodicity in the attacks, though this is not always well marked. In gout there is generally some slight lesion of the heart detectable, and in any case we must wait ulterior development. In syphilis there are usually other symptoms present, and probably a definite history of infection. In early middle life intercostal neuralgia may occasionally be found connected with spinal disease, and in one such case that came before me some years ago, about a couple of years elapsed before the disease unequivocally declared itself, and the true nature of the case was revealed.

In chlorotic women, or in weakly anæmic individuals of either sex, pain beneath the left mamma is very common, and when severe it is sometimes complained of as a heart pain and mistaken for angina. Usually this pain is an intercostal neuralgia and may be recognised by the three painful points, one over the intervertebral foramen, another about the middle of the intercostal space, and the third close to the sternum. Of these the first is the best marked and the one most seldom absent. Very often, however, this infra-mammary pain is a myalgia, and is recognised by the usual



phenomena, limitation in extent, aggravation on movement, and tenderness on pressure, the tender points corresponding to the tendinous origins and insertions of the muscles. In myalgia the pain is limited and does not radiate into the arms or along other nerves; in intercostal neuralgia this is not absolutely nor uniformly the case; radiation of the pain is always a suspicious symptom.

Constipation dependent on torpor of the colon is sometimes accompanied by neuralgic pains radiating from the neighbourhood of the *scrobiculus cordis* over the edge of the false ribs, and occasionally into the cardiac area itself. The pain in such cases is constant, with occasional exacerbations; it always radiates from some part of the colon, and may extend round the chest, or into the cardiac area, but it does not shoot upwards, or down either arm; it is neither excited nor increased by exertion, and it does not get worse through the night. If associated with spanæmia the heart may have all the usual murmurs, but the pulse is always soft and compressible.

Torpor and congestion of the liver, so constant an accompaniment of gastro-duodenal dyspepsia, is often associated with pain below either clavicle about the second interspace. This probably arises from irritation of the phrenic nerve radiating as pain into the upper intercostal nerves. On the right side this simulates lung disease; on the left it is often mistaken for a heart pain.

The pain of acute commencing pleurisy—if any way near the cardiac area—is sometimes mistaken for a heart pain, especially at first, when the respiratory movements are restricted by the acuteness of the pain, and no friction is to be heard. In cases of this character the thermometer helps to keep us right.

The heart itself often suffers from acute stabbing, cutting, burning, or stinging pains, unaffected by exertion and unaccompanied by any other sign or symptom of cardiac disease. In early life these pains are probably rheumatic in character

and may pass away without serious detriment, though at times they end in pericardial adhesions or possibly in stenosis of the mitral opening, but not without the concomitance of other symptoms which help us to recognise the true nature of the case. In later life the pains described are gouty in character, and frequently terminate in angina.

Finally, there is a cardiac pain dependent upon pressure on the cardiac nerves. This may arise from a tumour too small to be detected by physical exploration, and then it is apt to be disparaged as a mere neurotic pain of no importance. Should the pain be caused by the pressure of an enlarged gland, the danger may be but slight and must depend mainly on the cause of the enlargement. But should the pressure be caused by a small substernal aneurysm, the danger is great and imminent. In a few cases it is possible to make a fairly accurate diagnosis of such an aneurysm, but in many this seems impossible. In all we must exercise the greatest care and circumspection in regard to diagnosis, and in spite of the largest experience, an error may be readily committed; for the patient's sake it is better to err in excess of caution. But though substernal pain is often the sole symptom of a small aneurysm or tumour in this situation, this is sometimes combined with more evident symptoms of angina, and is occasionally associated with compression of the anabolic nerve (the vagus), and is then accompanied with tachycardia, depression of the dynamic force of the nerves implicated lying at the root of both phenomena.

Angina consists of a congeries of symptoms in which pain usually predominates, and is associated with a sense of impending dissolution, always present, but most intense in the severer forms; moreover, as Latham has said, these symptoms may be "incident to any form of organic disease of the heart, but they are constant to none."

The pain of angina varies from a dull agonising ache to an excruciating agony that, starting from the region of mid-sternum, shoots upwards to the left, and sometimes into both

shoulder joints, extending down to the elbow or along the ulnar nerve to the fourth and third fingers of the left hand and sometimes of both hands. Occasionally the pain shoots up the neck, generally on the left side, or into the *scrobiculus cordis*; more rarely the pain shoots into the loins or down the legs. The sufferer is said to have a feeling of suffocation; so far as I have been able to judge in the attacks I have observed, the feeling is rather one of intense anxiety lest the slightest movement should precipitate the end which seems so terribly near. The breathing is perfectly free. The countenance may be pinched, ghastly pale, and covered with beads of perspiration (*facies Hippocratica*). More often the face is little changed, save only for an anxious, haggard expression. By the time we get to the patient the pulse is intermittent, feeble, or irregular; but it is sometimes, when the angina is uncomplicated by valvular mischief, quite regular throughout the whole of the paroxysm. After a few minutes, or more rarely within an hour or two, the pain ceases as suddenly as it came, and the patient finds himself as he was, puzzled to know what has happened to him, and scared at the prospect of a renewal of the attack. There is nothing pulmonary in this seizure; the air enters freely into the lungs if the patient has the courage to breathe, and full inflation of the lungs has no effect on the duration or character of the attack. It has no connection with spasmodic asthma, nor with ordinary cardiac breathlessness, but in one of its forms painless breathlessness is a prominent symptom, and has received the name of cardiac asthma. Angina does not arise from cardiac strain, or at least from any immediate or recognisable cause of strain. Some trifling exertion or some emotional excitement of the heart's action are the most usual excitants of an anginous seizure, but in Arnold's case, as in some others, even the primary attack is devoid of this provocative, the patient waking from sleep in a paroxysm of anginous pain. In most cases it is not till after several and often numerous attacks, brought on by trifling exertions, that

the disease reaches such a height as to come on when the patient is at rest or asleep. In cases of this character the readiness with which the attack is brought on seems an indication of increased myocardiac failure; when it primarily occurs at night, as in so-called cardiac asthma, it apparently depends upon a difference in the exciting cause.

Should death occur during a paroxysm the heart may be found contracted and empty, or it may be uncontracted, flabby, and full of blood.<sup>1</sup> In all the cases I have seen die in a paroxysm, death has always been preceded by gradual failure of the pulse, ingravescent asystole—a mode of death which may occupy a varying time, from half an hour to a week or more;<sup>2</sup> it leaves the heart, as a rule, in diastole after death, and is unattended by any feeling of faintness, only by a sensation of impending dissolution, if there be anything beyond apathy present at all. A considerable number of sufferers from angina die without pain at the last, from *angina sine dolore*; they do not faint, the heart simply fails to contract and they die from sudden and complete asystole.<sup>3</sup>

The lesions found after death from angina vary in each case, but they are always of a similar character, one that indicates serious impairment, often long continued, of the metabolism of the myocardium. Atheromatous coronaries are common enough, and by some have been regarded as the true cause of the paroxysms; but sclerosis of the coronaries is too often met with where there has been no antecedent angina to permit of their concurrence being regarded as other than accidental, or, if causative at all, then only remotely so.

Fatty degeneration of the myocardium is not infrequently found when angina has been present during life, and by some it has been looked upon as the cause of the attacks. But a fatty myocardium is often enough found after death when there has been no preceding angina, so that it also cannot be

<sup>1</sup> Vide Quain's *Dictionary of Medicine* (1894), vol. i. p. 77.

<sup>2</sup> Vide p. 3; also *The Senile Heart*, p. 138, etc.

<sup>3</sup> Vide *The Senile Heart*, p. 145, etc.



regarded as anything but an accidental concomitant.<sup>1</sup> But fatty degeneration we know to be due to faulty metabolism, the result of local disturbance of the circulation,<sup>1</sup> and in the myocardium this is more often due to sclerosis of the coronaries than to any other cause. These two lesions are thus linked together as cause and effect, and when they are concomitant with angina the sclerosed coronaries may be regarded as the ultimate cause of that symptom. Faulty metabolism is indeed the one factor common to every condition of heart found in connection with angina. Sometimes this arises from the condition of the vessels, from diminution of the calibre of the coronaries at their origin at the root of the aorta or in any part of their course throughout the heart by embolism, thrombosis, or by inflammatory, atheromatous, or syphilitic processes. Rarely, as in Arnold's case,<sup>2</sup> the heart is long imperfectly nourished by arteries congenitally defective in size or number. More often the heart becomes enlarged—dilated and hypertrophied beyond the feeding powers of coronaries otherwise normal—though in this case failure in the nutritive qualities of the blood plays no unimportant part. Spanæmia or toxæmia—mainly from gout or malaria—have also an important effect in disturbing the metabolism of the tissues, and in this way are frequent causes of angina.<sup>3</sup>

The cardiac movements are primordial in character,<sup>4</sup> and, so long as metabolism is perfect, the heart is not only able to maintain these movements in all their primitive energy but also to store up a reserve so great that they continue to

<sup>1</sup> Thoma's *Pathology*, by Bruce (A. & C. Black : London, 1896), p. 415.

<sup>2</sup> Dr. Arnold died at the age of forty-seven of his first attack. "The heart was rather large. . . . The muscular structure of the heart was in every part remarkably thin, soft, and loose in its texture. The walls of the right ventricle were specially thin, in some parts not much thicker than the aorta. . . . Its cavity was large. The walls of the left ventricle, too, were much thinner and softer than natural, and the muscular fibres of the heart generally were pale and brown. . . . There was but one coronary artery, and, considering the size of the heart, it appeared to be of small dimensions."—Latham, *Diseases of the Heart* (London, 1846), vol. ii. p. 377.

<sup>3</sup> Thoma, *op. cit.* p. 415.

<sup>4</sup> Foster's *Textbook of Physiology*, 5th edition (1888), p. 288 *et antea*.

go on, in some animals, for hours after the organ has been severed from its organism. This large reserve of energy enables the heart to carry on its work with apparent efficiency in spite of long-continued untoward influences, the result of a nutritive pabulum deficient in quantity or defective in quality. Sooner or later, however, imperfect metabolism tells, the energy of the myocardium fails, and it comes more readily under the influence of those agencies which, acting reflexly through the nervous system, dominate the rate and regularity of its movements. Ultimately the myocardium becomes so imperfect that though the heart gets along fairly well with its ordinary work, yet when called upon for the slightest extra exertion it either fails to respond and sudden death ensues, or its imperfect action is accompanied by pain more or less severe, which shoots along the course of those sensitive nerves with which the sympathetic or katabolic nerve is embryologically connected.<sup>1</sup> This is angina pectoris. Angina is a nerve pain, a neuralgia of the heart; but it is something more—it is a neuralgia only developed on a sudden call for exertion. It differs from ordinary neuralgia in the neighbourhood of the heart in that it is always based upon imperfect metabolism of the myocardium, generally of long continuance; that it invariably darts along the course of certain special sensitive nerves, and that it is only developed as the result of a disability on the part of the myocardium to continue its function, generally brought to a crisis by some sudden call for exertion on the part of the heart. The pain of angina may not be exactly due to ischæmia, but it is closely allied to pain arising from that cause; to those atrocious pains that attend compression of an artery for aneurysm, especially at the moment the vessel becomes completely occluded; to those pains due to a similar cause that precede the appearance of senile gangrene, as well as to those which precede, accompany, and follow attacks of local asphyxia (Raynaud's

<sup>1</sup> Vide Gaskell, *The Journal of Physiology*, vol. vii. p. 1, and especially pp. 41 and 46.

disease). That is to say, angina is a pain—a neuralgia—similar to that which affects sensitive tissues in which the discharge of function is hindered by defective metabolism.

Whenever, from any emotional cause, or from the slight exertion of ascending an acclivity, or from a sudden rise of blood-pressure from reflex or other causes, the augmentor nerve has to make a call upon the heart for increased action, to which it is unable to respond because of long-continued imperfect metabolism from one or other of the causes just referred to, the call for increased katabolic action is at once followed by exhaustion revealed as an agonising pain shooting along some or all of those sensitive spinal nerves with which the katabolic nerve is embryologically connected.

As causes of that imperfect metabolism to which an anginous attack is primarily due, there are, besides those vascular changes which give rise to a more or less positive ischæmia, many causes that impair metabolism by a qualitative ischæmia, the defect being in the quality and not in the quantity of the nutritive fluid. These conditions may be classed as toxæmic causes of angina; chief among them we have amongst the young—for angina and even fatal angina is not confined to the old<sup>1</sup>—a spanæmic condition of the blood; amongst those past middle life, gout; and amongst those of all ages, malaria. Tobacco, tea, alcohol, and other poisons have often been spoken of as toxæmic causes of angina. I have seen many cases of severe palpitation and cardiac irregularity from these causes, but never angina, the nearest approach to this being a slight pain accompanying the action of the augmentor nerve after a bad bout of irregularity. As I have seen tobacco used very freely, in one case fifteen large and strong cigars, and in another fifty cigarettes inhaled daily, both producing about a maximum of cardiac discomfort but no angina, I am inclined to believe that in those cases of tobacco angina recorded some other cause must have contributed to its production, and the most probable contributory

<sup>1</sup> Vide *The Senile Heart*, pp. 115, 123, etc.

cause seems to be imperfect metabolism due to spanæmic blood<sup>1</sup> from gastric disturbance. Malaria is not an uncommon cause of angina; of this I have seen several well-marked cases yielding to antimalarious treatment. In them there is always the history of exposure to the cause, more or less of malarious spanæmia, and more or less distinct periodicity in the attacks. Malaria is a well-known cause of neuralgia which may affect the nerves in the neighbourhood of the heart, but anginous symptoms do not arise until the cardiac metabolism has become affected. The same remark may be made of gout: gout is a common cause of neuralgia and often affects the nerves in the neighbourhood of the heart, but anginous symptoms do not arise until cardiac metabolism has been seriously interfered with. In both classes of cases there is generally some exciting cause traceable, some exposure to cold, some trifling exertion, or some error in diet.

I have elsewhere described<sup>2</sup> a case of cardiac neuralgia for long under observation, and by some regarded as angina. The exact nature of this case was always obscure until she one day told me—after twelve years' observation—that she had recently been subject to occasional pain in her wrists and fingers accompanied by great irritability of temper, and that at these times her cardiac neuralgia completely disappeared, only to return so soon as her gouty joint pains left her. As yet this patient's heart is quite unaffected, though she is over fifty; by and by, as years creep on, the cardiac metabolism is sure to become impaired, and then the cardiac neuralgia will probably pass into true angina. Similar cases are not uncommon. So long as the gout affects the nerves alone and there is only neuralgia, a fit of regular gout may afford relief, but when the cardiac metabolism gets impaired

<sup>1</sup> As I write this I am confronted by a man of fifty who has suffered from bad angina for the last two months, for which he blames tobacco. But his heart is dilated, and his blood spanæmic; besides, he has been in the habit of smoking but little over 2 oz. of strong tobacco per week. This case supports my view on above details.

<sup>2</sup> Vide *The Senile Heart*, p. 119.



and the attacks are truly anginous, then a fit of true gout gives but slight if any relief, and rather tends to hasten the end.

Angina is sometimes feigned, and is occasionally of a purely hysterical character, but no man of any experience is likely to be deceived by either form, provided he has an opportunity of observing an attack. Mrs. Chisholm, whose case was formerly commented upon,<sup>1</sup> was an example of the former character, and a very clever impostor she was, giving a great deal of trouble before she was detected. Some years ago we had in the case of the girl L. a well-marked instance of the hysterical variety. L. suffered from aortic regurgitation, from which she subsequently died, and the true nature of her seizures was not at first suspected. One day, however, I happened to find her in one of her attacks at visit; I at once recognised its hysterical character, and took measures to prevent a recurrence, which were happily successful. I have seen several other cases of hysteria simulating angina, but none so well marked as this one, and, let me add, never any one in which there was less cause for suspecting hysteria, or more apparent reason for believing the paroxysms to be real.<sup>2</sup>

Whenever we have cardiac uneasiness or irregularity after middle life associated with early indications of a weakened myocardium, an auricular murmur, an accentuated pulmonary second, a blunt first sound, or a transference of the impulse from the left to the right apex, we should be exceedingly guarded in our prognosis. These are not cases of angina, but they belong to the same category; the symptoms depend upon failure of cardiac metabolism, and in the most unlikely cases may terminate suddenly in asystole, death from *angina sine dolore*.

Cardiac asthma is another variety of *angina sine dolore*; it too is an indication of failure in the cardiac metabolism and

<sup>1</sup> *Vide* first edition of this work (London, 1876), p. 279.

<sup>2</sup> M. L., admitted to Ward XXXIII. on 28th January 1880; died from sudden asystole, 31st May 1880.

is often the earliest indication of the approaching end. We know that cold is a common cause of angina by inducing contraction of the superficial vessels, raising the blood-pressure and thus increasing the work of the heart, a form of vaso-motor angina.<sup>1</sup> But a similar contraction of the arterioles generally is often produced by some organic derangement—stomach, liver, etc.—or by some impurity of the blood, then we have coldness or numbness of one or more of the extremities followed immediately by an attack of angina. In one patient the chilliness was at first limited to the right arm alone, and his heart at first was fairly good, but this organ dilated considerably before his death, which happened suddenly about two years after he was first seen.

Now and then towards the close of life the attacks of painful angina cease, and the patient suffers from attacks of breathlessness only; at other times attacks of angina and of cardiac asthma alternate, and at still other times an attack commencing as angina terminates in cardiac asthma. I myself have assisted at the development of an attack of cardiac asthma of the latter variety. A man aged fifty-seven had suffered from angina for years; the attack was brought on by exertion or by emotion, and the pain shot from midsternum through to the back and down the left arm, occasionally down the left leg, and sometimes down the right arm also. The heart had a feeble impulse; its action was slightly irregular, the aortic second was accentuated, and the first sound over the apex was blunt; the radial pulse was tense—signs which indicated a high blood-pressure, and a dilatable and probably somewhat dilated heart. As I listened to the heart-sounds, nervous excitement brought on an attack of angina, pain accompanied by a feeling of suffocation. Gradually a distinct auricular murmur was heard to develop, indicating regurgitation through the mitral valve from ventricular dilatation (*vide antea*, p. 170), and *pari passu* with this the pulmonary second not only became markedly accentuated but also

<sup>1</sup> *The Senile Heart*, p. 131.

acquired a distinctly booming quality. Residual accumulation, due to irregular and imperfect ventricular action, had overdistended the ventricle, promoted regurgitation through the mitral valve, and had thereby caused considerable congestion of the lungs. As a consequence of this transference of the blood-pressure from the aortic to the pulmonary system the radial pulse, which had been full and tense, became gradually small and feeble, and even while the patient was at rest there was an increased breathlessness that amounted to a well-marked though slight attack of cardiac asthma.

Doubtless ordinary attacks of cardiac asthma have a similar origin. A reflex spasm of the systemic arterioles suffices to induce—without pain—residual accumulation and overdistention of the left ventricle, with its natural results—mitral regurgitation, pulmonary congestion, and breathlessness.

There is every reason to suppose that the arterial spasm, which is so evidently the cause of local asphyxia (Raynaud's disease), and which is so important an agent in inducing an attack of *angina vaso-motoria*, occasionally invades the heart itself, either as part of a general condition, or it may be as a distinctly local affection, and in this way may very possibly be a cause of angina in cases where no other seems obvious.

In angina pectoris, as in other neuralgiæ, we have a permanent lesion coupled with only occasional attacks.<sup>1</sup> With a large experience of every variety of angina, I can safely say that I have never yet seen a case of angina in which I was unable to detect some of the physical signs of cardiac dilatation, some marked indication of imperfect cardiac metabolism; and I may also say that the less there seems to be the matter with the heart, the more grave is the prognosis, if the attacks be at all serious. From my point

<sup>1</sup> In regard to the periodicity frequently presented by neuralgiæ depending upon grave organic lesions, *vide* Trousseau's *Clinical Medicine*, New Sydenham Society edition, vol. i. p. 598.

of view this is readily understood, for if we regard angina as essentially due to imperfect metabolism, and that it has only recently come on in a dilated heart, there is always the hope that this may be remediable, so far, at least, as to enable us to get rid of the angina. But if we find angina implicating an apparently healthy heart, we at once recognise that the hindrance to the cardiac metabolism is of an acute character and is most probably due to some interference with the coronary circulation, which we cannot hope successfully to influence. Even these serious cases do not always die immediately; some of the most serious I have seen have lived a couple of years, but life was one continuous suffering, and the heart that at first had nothing detectable wrong with it showed towards the end gradually increasing signs of dilatation.

I have already pointed out that after middle life cardiac irregularity is not always free from danger and may quite suddenly terminate in death from *angina sine dolore*, but even in early life tumultuous action of the heart may terminate in fatal angina.

CASE XXX. A. S., a male, aged twenty-four, admitted to Ward V. 6th October 1877, complaining of palpitation and pain in the præcordial region. The patient stated that he had suffered in this way for fourteen weeks, and that the pain was sometimes a mere uneasiness, and at others more acute. On admission the patient presented a somewhat anxious expression; he was well nourished and all his organs healthy except the heart. The heart beat rapidly (120 per minute) and in a somewhat tumultuous manner; the radial pulse was quite regular. On auscultation the first sound over the apex was impure; the pulmonary second was markedly accentuated, and a faint diastolic murmur was audible over the aorta at midsternum. The case was at once recognised as a serious inflammatory affection of the heart or ascending aorta, the exact seat of which could not be localised on account of the absence of any distinctive symptoms. The treatment



consisted in the administration of full doses (15 grains) of iodide of potass in a bitter infusion three times a day, with perfect rest in bed, and an unstimulating diet. Under this treatment his heart quieted down, but the pain increased and became localised as a constant pain in the *scrobiculus cordis*, unaffected by pressure. This extension of the pain to the epigastrium was at first regarded as due to the action of the iodide on the stomach; but as the pain recurred in a distinctly paroxysmal manner, and as the pulse became rapid and feeble both during the paroxysm and for some little time after, I ventured to complete the diagnosis and to state that in all probability there was an acute end-aortitis implicating the origin of the aorta and the orifices of the coronary arteries. The concomitant dilatation of the heart was undoubtedly due to febrile relaxation of the tissues, favoured by imperfect metabolism, the result of the obstacle to the coronary circulation. At first inhalations of nitrite of amyl gave great relief, the paroxysms lasting from five to fifteen minutes. But in the final attack, which commenced about four o'clock on the morning of 24th October and lasted for about two hours, the amyl was of no use and the only relief from the intense agony was obtained by chloroform inhalations.

At the autopsy on 25th October the body was found to be well formed and fairly muscular. *Rigor mortis* and *post-mortem* lividity were well marked. There were about 6 oz. of clear serum in each pleural sac, and about 2 oz. of a similar fluid in the pericardium. The blood was remarkably fluid. The heart weighed 13 oz.; the ventricular cavities were slightly dilated, and their walls slightly hypertrophied. The mitral orifice was enlarged, admitting four fingers; cone diameter, 1.6. The cusps were natural. The tricuspid orifice was also enlarged, admitting six fingers; cone diameter, 1.9. In the wall of the aorta, just beyond the aortic cusps, there was a ring of atheromatous thickening which involved the whole circumference of the vessel at and a little beyond

the sinuses of Valsalva. In this situation the *tunica intima* was swollen to twice or thrice its normal thickness, and presented a clear, translucent aspect, broken here and there by points of fatty degeneration. The openings of the two coronary arteries lay in the midst of this atheromatous area, and were both so extremely contracted as barely to admit the point of an ordinary surgical probe. The aortic valve allowed water to leak through it slightly, but it might be said to be practically competent. The cusps were slightly thickened at their free margins, and above the *corpora aurantii*. The muscular substance of the heart was everywhere of good colour and consistence, and on microscopic examination it presented no abnormality except the presence of a considerable number of reddish-brown pigment granules in some of the fibres. All the other organs of the body were perfectly healthy, but somewhat congested.<sup>1</sup>

In this most interesting and probably unique case you will observe that we have had a fatal angina with a practically healthy heart. The slight dilatation, slight hypertrophy, and trifling leakage through two orifices, were nothing more than we may find in any spanæmic heart, especially when febrile excitement co-exists. Similar conditions are to be found in hundreds who make perfectly good recoveries. The fatal lesion was evidently the blocking of the coronaries; this was what we found, and this was precisely the lesion predicted to be the most probable one. This opinion was based on the obviously casual connection between the acute attack and the angina, and on the fact that in by far the larger number of cases angina seems to depend upon some interference with the blood supply to the walls of the heart itself—to the myocardium. Of this the case in point is an admirable and instructive example, and the celebrated case of Dr. Arnold is no less so. For though his coronary artery—

<sup>1</sup> Condensed from the *Pathological Records of the Edinburgh Royal Infirmary*. A chromolithograph of this heart forms the frontispiece to this volume.

he had but one—was neither diseased nor obstructed, it is stated that, “considering the size of the heart, it appeared to be of small dimensions, and with some difficulty admitted a small director” (Latham, *loc. cit.*); while the thin, soft, flabby texture of the heart sufficiently testified to the inadequacy of its feeding power.

Acute inflammatory angina, as in the case just narrated, is certainly a rare affection, and not less so is acute traumatic angina, of which the case now to be described is the only example that has come before me.

CASE XXXI. J. L., a married woman, aged fifty, stout, healthy, and who had passed through her life without an ache or a pain except those incident to maternity, slipped and fell on the street in the beginning of January. Being rather heavy she was considerably shaken, but apparently not otherwise injured. By and by, however, paroxysms of angina set in, and continued gradually to increase in severity. There was nothing abnormal to be detected about her heart, but she died suddenly in a paroxysm about the middle of March of the same year. Unfortunately I was unable to obtain a *post-mortem* examination; but from the sudden onset of the angina, its obvious dependence on some injury received at the time of falling, and the general resemblance of the most prominent symptoms to those of substernal aneurysm, there is every reason to believe that they were due to some such cause. Probably the middle coat of the aorta was fissured transversely at the time of the fall just above its cardiac origin, and the angina was due to interference with the coronary circulation, and possibly also to pressure on some of the branches of the cardiac plexus by the gradually increasing aneurysm thus formed, while death was probably caused by this aneurysm bursting into the pericardium.

The two cases just narrated both died during a paroxysm. Judging from my own experience by far the larger number of fatal seizures are apparently painless. Death happens

precisely as it does to animals which have had their coronary arteries artificially blocked. Sometimes the heart fails suddenly—*angina sine dolore*—often after a longer or shorter period of freedom from pain. At other times complete failure is preceded by a longer or shorter period of ingravescent asystole (more or less conscious sinking). The pre-existing state of cardiac metabolism, and the nature of the exciting cause, have all a special influence upon the mode of dying. It is only rarely that patients who have suffered from serious angina die from asthenia and gradually increasing dropsy, but this sometimes happens. One old friend, who for several years suffered from many comparatively slight attacks of angina, lunched cheerfully with some friends, walked with apparent ease to a railway station but a short distance off, sat down, and died. Others having suffered more or less severely for years have died suddenly without giving any indication of suffering.<sup>1</sup> One well-known literary man I myself saw die from ingravescent asystole about a week after his last paroxysm. He had long suffered from angina, with all the signs of a weak, dilated heart, due, as was subsequently discovered, to atheromatous coronaries, and I had brought him safely through two most severe attacks with a comparatively painless interval of a year between them. Subsequent to the last attack he had been confined to bed with symptoms of pulmonary oedema, a common result of a severe paroxysm. When apparently convalescent he obtained permission from his medical attendant to rise from bed, and while dressing his weak heart failed, and ingravescent asystole set in. There was no pain. When I reached his apartment he said, "Doctor, this is very different from anything I have had before," and he died quietly after drinking about half a glass of brandy given him in the hope of stimulating the heart to more vigorous contraction. The whole act of dying occupied about half an

<sup>1</sup> Various illustrations of the modes of dying in similar cases are to be found in *The Senile Heart*, p. 133, and at p. 144.



hour. At the *post-mortem* examination "both coronaries were found atherosed and obstructed, and in the substance of the left ventricle there was an elongated patch of advanced fatty degeneration. The limits of the patch were well defined, and the appearance presented bore a considerable resemblance to that of a hæmorrhagic infarction which had undergone fatty degeneration."<sup>1</sup> The heart itself was somewhat dilated.

When a spanæmic heart is called upon for extra exertion the impoverished or imperfectly oxygenated blood has to be sent through the myocardium much oftener per minute than healthy blood, that sufficient pabulum may be provided for the muscular metabolism. To provide for this the augmentor nerve is called into action and the heart-beat becomes rapid and forcible. Now and then, even when defective metabolism has gone so far as to cause pain (angina) to be associated with any attempt to respond to a call for increased action, some are able to disregard this warning, and by a voluntary effort force the heart into stronger action, flush the myocardium with an increased blood supply and thus overcome the breast pang. Perhaps this happens more frequently when the angina is an accompaniment of aortic regurgitation than under any other circumstances. I have already narrated one such case (2nd ed. p. 273), and I can recall several others, but the experiment is dangerous and failure sudden death.<sup>2</sup>

In the treatment of angina our immediate predecessors were sadly handicapped; they had nothing to trust to but the external application of cutaneous irritants, and the exhibition of stimulants and narcotics by the mouth—a vain hope when moments are precious, and to the time needed for absorption there has to be added the further

<sup>1</sup> I quote from a letter received from Dr. John Wyllie, at that time pathologist to the Royal Infirmary, who made the dissection, the full report having been unfortunately lost. The specimen is in the University Museum.

<sup>2</sup> Forbes, *Cyclopædia of Practical Medicine*, vol. i. p. 94, mentions similar cases.

delay of that act caused by a failing circulation. Modern discoveries have altered all this; by inhalation we can thoroughly narcotise a patient in a few seconds, and by means of a hypodermic injection we can secure within a few minutes a painless unconsciousness which will last for many hours.

In determining how best to relieve the pain of angina we must duly consider its cause, incapacity for action on the part of the myocardium, the result of long-continued defective metabolism, or more rarely of sudden and more or less complete ischæmia. The state of the myocardium seems to be similar to that of the leg muscles when the veins are varicose or blocked. A sensation of weight, fatigue, and sometimes pain, on taking exercise, suddenly compels the sufferer to call a halt when the symptoms pass away. But the heart cannot halt; it must either cease to act, or its action continues feeble, hampered, and accompanied by pain and by all those inexplicable sensations that make up the syndrome of angina. The initiative of such an attack is always some sudden call for extra exertion on the part of the heart; of this I think there can be no doubt; but the question then occurs, Is this initiative accompanied by any rise in the blood-pressure, by any increase of the intra-arterial blood-tension? This was a theory originally started by Dr. Lauder Brunton, and it must be confessed that many of the conditions that accompany the circumstances that initiate an attack are of such a character as most likely to give rise to an increase in the blood-pressure. Accordingly, when seen early, there are probably but few cases in which an initial rise of blood-pressure is not to be detected, but this does not last long, and the pulse speedily becomes small, feeble, and irregular, a state of pulse with which those accustomed to see cases of angina are probably much better acquainted than with the firm hard pulse of high tension. The belief that an attack is always initiated by a rise in the blood-pressure has led to the employment of various vascular stimulants as means of affording immediate

relief, and this certainly frequently follows their use, but whether *propter* or only *quia post* is perhaps not always very easy to determine. The remedies of this class most frequently employed are the nitrite of amyl and nitro-glycerine; the former is given by inhalation and acts very quickly; the latter is given by the mouth, either in the fluid form, when its action is almost as rapid as that of amyl, or in the form of a lozenge, when it acts somewhat slower, but this is counterbalanced by convenience of carriage. These are indispensable remedies; the patient can readily carry them about, and relief generally follows their employment, but not always, especially in serious cases, even when used most freely. I have known a pocket-handkerchief soaked in nitrite of amyl laid over the mouth and nose and the amyl freely inhaled and followed by all the usual symptoms without much relief; and I have also seen as much as ten minims of a one per cent solution of nitro-glycerine taken without any relief at all. In all cases of angina the routine treatment is to prescribe nitrite of amyl, usually in glass capsules, several of which may be crushed in a pocket-handkerchief and their contents inhaled, or, what I prefer, nitro-glycerine in doses commencing with one-hundredth of a grain. In a few cases the relief obtained is sudden and complete; more often the relief that follows is gradual and doubtfully due to the treatment, while in a few cases no relief seems to follow even the most prodigal use of these drugs. Then we are forced to have recourse to the free administration of chloroform, and this must be given so freely as to narcotise the patient rapidly and completely; given in this way I have not yet seen any case that has not been relieved, though I have seen several in which the relief was not permanent enough to restore the patient to comfort. In these cases the chloroform has had to be supplemented by the hypodermic use of morphine, and of this I have never hesitated to give a sufficient dose, generally from half a grain to a whole grain; such a dose as this has kept the patient asleep for some hours and he woke

free from pain but exhausted. In treating any case of angina we cannot shut our eyes to the serious nature of the affection, and that death may possibly occur even during our treatment; this is unpleasant, but, while not forgetting the possibility of such an occurrence, we are bound to run every risk for the sake of relieving suffering so intense. I myself have been fortunate enough never to have seen any untoward result. Anstie had a dread of chloroform in such cases and says that "the effect of a *powerfully* charged atmosphere, breathed only once or twice even, would be instantaneously fatal."<sup>1</sup> I well remember one case of extreme cardiac pain and dyspnœa in a patient almost moribund from disease of her heart, and who did die only a few days subsequently, yet in her the immediate effect of chloroform inhalation was to restore the pulse to her radial arteries. In a few seconds, instead of being pulseless, black in the face, and gasping for breath, partly from pain, and partly from extreme dyspnœa, she began to breathe freely, became quiet and natural in appearance, and in a short time she was able to lie down and rest quietly. But you may say that in angina the heart is always flabby and ill-nourished and often fatty; is chloroform not dangerous in cases of fatty heart? The next case I shall relate gives the best possible answer to such a query as this. But I must first most emphatically say that I doubt the possibility of diagnosing a fatty heart. We may suspect its existence, because the physical signs seem to warrant the supposition, and the conditions present seem favourable to its development, but little acquaintance with morbid anatomy is, however, requisite to teach us that fatty degeneration of the myocardium is not always present even in apparently the most likely conditions, and that it is not infrequently discovered where it has been wholly unsuspected.<sup>2</sup> I may

<sup>1</sup> *Op. cit.* p. 80.

<sup>2</sup> While this is passing through the press, this statement has received a most forcible illustration in the death, from rupture of a fatty heart, of one of our best-known and most esteemed medical practitioners, whose healthy appearance and great vigour both of body and mind were entirely opposed to



refer to the case of Bridget Henry, who died from chloroform in the Cincinnati Hospital, U.S.A., on 13th October 1870, as a well-marked example of a fatty heart with a normal impulse.<sup>1</sup> But indeed of this there could be no more striking instance than that of Mrs. Tait, which I shall presently relate to you. The rapid and powerful action of chloroform makes it a dangerous agent in incautious hands, and an anæmic condition renders the heart more sensitive to its action and more liable to be fatally overpowered by an overdose. But I know of no state of the heart which should deter us from its cautious but effective employment when that is otherwise indicated, as it imperatively is in certain cases of angina pectoris.<sup>2</sup> Ether has long been used for a similar purpose, the chief objection to its employment being that its action is not rapid enough. Chloroform acts much quicker, even more effectually, and is perfectly safe. If we put some chloroform in the bottom of a tumbler, or provide the patient with a chloroform smelling bottle, he may safely enough be entrusted to employ the drug unaided and so save much time. The moment narcosis occurs the smelling bottle drops, and with it rolls away all risk of an overdose.

CASE XXXII. Mrs. Tait, long a nurse in the Edinburgh Infirmary, died in Ward XIII. on 30th March 1871.<sup>3</sup> She was over eighty years of age, and had long suffered from a dilated heart and from angina, the paroxysms of which were very severe with but short intermissions. For the last few weeks of her life she was almost constantly under the in-

all the more generally received ideas in regard to the indications of this form of degeneration.—Note in second edition (1876); *vide* also note, p. 367.

<sup>1</sup> *Chloroform Deaths*, by W. W. Dawson, M.D., surgeon to the Cincinnati Hospital, 1871, printed by Robert Clarke and Co., Cincinnati, U.S.A.

<sup>2</sup> In his work on *The Bearings of Chronic Diseases of the Heart upon Pregnancy, Parturition, and Childbed* (London, 1878), Dr. Macdonald says, *apropos* of a primipara labouring under aortic insufficiency: "The patient looked pale . . . and complained that she felt ready to faint with every pain." But when chloroform was given "the pulse became stronger and steadier, instead of feebler and more irregular, as I feared it might," *vide* p. 147.

<sup>3</sup> Margaret Tait, admitted 3rd January, died 30th March 1871.

fluence of chloroform or morphine, or of both, the morphine being injected hypodermically so soon as the chloroform narcosis was fully established, so that its soothing influence might continue when that of the chloroform had passed away. She died at last, not suddenly, but gradually, from asthenia, worn out by her age and sufferings. After death the aorta was found dilated, the orifice of the middle coronary artery—there were three in her heart—almost entirely blocked by atheromatous deposit. Her heart was not only thin-walled and somewhat dilated, but of a pale yellowish tint, soft, and thoroughly fatty. The muscular fibres were completely degenerated—none could be more so—yet chloroform produced in her no dangerous symptoms, and, far from shortening her life, it was credited with prolonging it, and it certainly made her life more endurable. I may add that the apex beat continued firm to the last.

But however satisfactory our treatment of a paroxysm may be, the treatment during the intermission is of much more importance, and is not infrequently attended by most brilliant results. This can only happen when the heart is simply dilated, feeble, and ill-nourished; it is obvious that no treatment can be of any permanent avail when we have to do with atheromatous disease of the coronaries so extensive as materially to interfere with the nutrition of the myocardium, with thrombosis of these vessels, or with any lesion that irremediably affects the circulation through the heart. Inasmuch, however, as the treatment of angina—apart from these conditions and from the presence of a paroxysm—differs in no respect from that of an ordinary feeble heart, consideration of it may be postponed till we speak of the treatment of cardiac disease generally (*vide* Lecture XIV.)

## LECTURE XIII

### ON PERICARDITIS, ENDOCARDITIS, MYOCARDITIS, AND CARDIAC HYPERTROPHY APART FROM VALVULAR DISEASE

THOUGH acute articular rheumatism is common enough with us, yet rheumatic pericarditis is an affection comparatively unknown. This is all the more remarkable as our largest ward is a female ward, and, as you are aware, rheumatic pericarditis is more prone to attack young and weakly persons, especially females, than any other class of patients. Were it not that rheumatic pericarditis is usually an early, and rarely a late complication—occasionally even preceding the joint affection—we might be inclined to attribute its rarity to the success of our treatment. But the fact that few of our patients come under treatment before the fourth or fifth day, and that nevertheless severe pericarditis is a rare affection, seems to indicate that serious rheumatic pericarditis is actually less common in Edinburgh than it is elsewhere, as it certainly seems to be less severe; and this is a probability which has also suggested itself to Dr., now Sir William Gairdner.<sup>1</sup>

As you are aware, the employment of salicin and the salicylates has completely revolutionised the treatment of acute rheumatism, so that six days is now rather a long period of treatment instead of six weeks, which was the former minimum. But neither salicin nor any of its derivatives is a true specific for this complaint; it undoubtedly shortens the febrile stage, but it does not appear to have any

<sup>1</sup> *Edinburgh Medical Journal* (January 1861), p. 630.

control over the occurrence of fatal hyperpyrexia, of which one remarkable instance occurred in a patient under my own care in Ward XV. This patient, a young servant girl, twenty-one years of age, was apparently progressing favourably under salicin treatment. Her temperature at night was only  $102^{\circ}$ ; in the early morning she seemed no worse and made no complaint, but at nine o'clock she was found to be unconscious, perspiring freely, with a temperature of  $107^{\circ}$ . She died at half-past twelve, with a temperature of  $111^{\circ}$  F., which still persisted one hour after death. On *post-mortem* examination no lesion was found to account for this remarkable and fatal hyperpyrexia, to which alone undoubtedly the death was due.<sup>1</sup> Neither has longer experience confirmed the hopes at first entertained that the shortening of the duration of the febrile stage would have an important effect in diminishing the risk of ulterior affections of the heart. Acute cardiac affections of rheumatic origin as a rule occur early in the disease, before the patient has come under treatment at all, and cannot well be prevented by anything short of an immunising treatment which would at once and rapidly extinguish the disease. This has still to be discovered. Meanwhile we may be very well content with the results obtained by the use of salicin. The contrast between the results thus obtained and those that followed former methods of treatment has been very well shown in Maclagan's recent work.<sup>2</sup> Certain drawbacks have been ascribed to the use of salicin and its compounds; one of these is that there is a great tendency to relapse, but the apparent relapse seems to be merely a recrudescence of the disease due to too early an omission of the drug, and can be readily prevented by continuing the treatment for a sufficient time after the acute

<sup>1</sup> *Vide* "Comments on a Case of Hyperpyrexia," by Theodore Cash, M.B., etc., *Edinburgh Medical Journal* (September 1878), p. 234.

<sup>2</sup> *Rheumatism, its Nature, Pathology and Treatment*, by T. J. Maclagan, M.D. (London: A. and C. Black, 1896), p. 202, etc., where are to be found several temperature charts under both the old and the new methods; the contrast is remarkable.



symptoms have disappeared, say for a week or ten days. In this way all risk of recrudescence is averted. The other drawback is a very real one; it is the production of toxic symptoms which may be serious or even fatal. These toxic symptoms are chiefly delirium, with or without increased rapidity, weakness, and irregularity of the heart and pulse. These toxic symptoms are chiefly found in connection with the use of salicyl compounds, and are said to be due to the employment of compounds of salicylic acid prepared from phenol. Whatever the cause, the danger is real, and salicyl compounds should never be employed when the pulse is rapid and the heart's action enfeebled.<sup>1</sup> On account of these toxic symptoms I myself prefer the use of pure salicin, which seems to be practically free from any toxic symptoms whatever. As Maclagan has told us, one ounce of salicin must be ingested before we can expect any marked benefit; the more rapidly, therefore, this quantity can be got into the system the sooner relief is obtained. Thirty grains may be safely given every hour till pain is relieved and the temperature begins to fall; afterwards the same quantity may be continued at longer intervals till the full ounce has been ingested. To complete the cure a second ounce must be given in similar doses, but at still longer intervals, and it is often advisable to give a third ounce in half-drachm doses three times a day, to ensure perfect and complete convalescence.<sup>2</sup> The patient must be clad in flannel and bedded in blankets, according to time-honoured custom in this Infirmary, which dates back to the days of Cullen,<sup>3</sup> and his joints must be wrapped in cotton-wool, and may have some anodyne liniment laid over them. The diet must be chiefly milk with small quantities of farinaceous food or chicken-tea if preferred, but beef and all its preparations ought to be

<sup>1</sup> Binz, *Lectures on Pharmacology*, New Sydenham Society edition, vol. ii. p. 271.

<sup>2</sup> Maclagan, *op. cit.* p. 191.

<sup>3</sup> *MS. Clinical Lectures*, in the Library of the Royal College of Physicians, Edinburgh.

rigidly excluded till long after convalescence has been fully established. I need not enter more fully into this matter at present; I have only mentioned it at all because though salicin and the salicylates fail to check rheumatic affections on the left side of the heart,<sup>1</sup> it is quite otherwise with rheumatic affections of the pericardium, which seem to be quite as amenable to salicin treatment as any other joint, and to require no other beyond some little help to soothe the pain and also to keep the joint itself as quiet as possible. If we take cardiac pain accompanied by slight but yet detectable effusion into the pericardium, or by friction at the base of the heart, accompanying acute rheumatism, as signs of rheumatic pericarditis, as they undoubtedly are, then I suppose the percentage of pericarditis occurring among rheumatic cases in the wards has been about the average, 25 to 30 per cent according to Bamberger, and of these the mortality has been *nil*. These cases were mostly slight and with very few exceptions the pericarditis was present on admission.

In not a few cases the rheumatic attack has been associated with cardiac disease of long standing, while in almost all the other cases we have been able to determine the existence of the auricular murmur, which, as I have already told you, is of such common occurrence in all febrile disorders, and depends upon a slight degree of cardiac dilatation.<sup>2</sup> Occasionally, though rarely, this has been accompanied by a murmur of similar origin audible in the mitral area. But the persistence of this mitral murmur, and of its cause, the dilated left ventricle, has been a very exceptional occurrence. Even when valvular lesion has subsequently developed, the restoration to health has been, as a rule, apparently complete, and the signs of valvular lesion have only slowly developed after some time, not less than a year, occasionally apart from any definite symptoms whatever; at times associated with repeated trifling rheumatic attacks of short duration, and at

<sup>1</sup> Binz, *op. cit.* vol. ii. p. 267.

<sup>2</sup> Lecture VI. p. 168.

other times with the persistence of chronic wandering rheumatic pains not always obviously affecting the heart. *Hæret lateri lethalis arundo* is an apothegm applicable to rheumatism in all its forms, and if it finds its most apposite application in rheumatic pericarditis, it finds it so rarely as to be of little consequence to the species, however important it may be to the individual himself. The following case is the most serious one of the kind that has occurred to me during fifteen years' connection with the Infirmary:—

CASE XXXIII.<sup>1</sup> David Davidson, a tailor, aged twenty-four, admitted to Ward V. on 16th November 1870, complaining of pain in the chest and limbs, difficulty of breathing, and dry cough. Patient said that he had always been healthy till about five years ago, when he began to suffer from slight rheumatic pains. The present attack commenced on 7th November with pains in the joints of his lower extremities, but he continued at work till the 10th. On the 12th pain set in about the heart, and on the 16th he came into hospital. Both his parents are dead; his father had been rheumatic, his mother not. Upon admission his expression was anxious; his skin felt hot and was soaked with sour-smelling perspiration. The temperature in the axilla was only 99·8°. He had pain in the right knee-joint, but it was neither swollen nor red. The patient felt a burning pain in the præcordial region; the cardiac percussion dulness was normal, but a loud, rough friction sound accompanied the movements of the heart; it was heard loudest at the base and with the systole, but it obscured both sounds. The pulse was 100, full, compressible, and markedly dicrotic, as is well shown in the accompanying sphygmogram (Fig. 28). The respirations were 40 per minute, and he had some cough with slight expectoration. The percussion note was clear over both lungs; the respiration was normal on the right side, but accompanied by coarse crepitations on the left. The patient's sleep was disturbed, and his appetite was defective; his tongue was coated, dry

<sup>1</sup> Case reported by Benjamin Tydd Hunter, clinical clerk.

and brown in the centre and towards the back, white at the edges. His bowels were normal. His urine was normal in quantity, acid, deep amber in colour, specific gravity 1026. It contained a floating cloud of urates, and gave with nitric acid a deposit of nitrate of urea; the chlorides were normal. The patient was put upon infusion of digitalis with nitrate of potass, and chloral at night. In two days the pulse had fallen to 96, and its diastolicism could no longer be detected. On 19th November the pulse had again risen to 100; the cardiac symptoms remained unchanged, but all the joints of the middle finger of the left hand and the left wrist were swollen red and painful, and his loins ached much. The patient was now put upon drachm doses of the tincture of the *actæa racemosa* every three hours, and a warm linseed

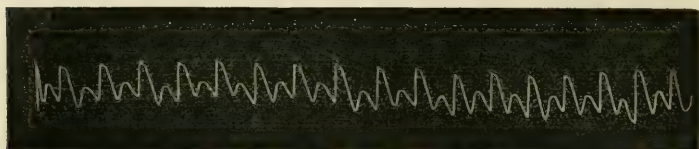


FIG. 28.

poultice was kept constantly applied over the cardiac region. On 22nd November the transverse dulness of the cardiac area at the level of the fourth rib on the left side measured six inches; the dulness did not rise above the third rib, and it was recognised as due to effusion within the pericardium by the disappearance of the apex beat, and by the dulness shifting as the patient was turned from one side to the other. On 24th November he had an attack of severe pleuritic pain, for which he had morphine injected hypodermically. This pain resulted in effusion into both pleuræ, the dulness extending as high as the eighth rib on both sides. His other symptoms moderated, and on account of the persistence of this effusion on 30th November he was ordered—

R Potasii iodidi,  
Potassæ bicarbonatis, aa ʒ ii.  
Infusi calumbæ, ʒ vi. Solve.

Signetur—One tablespoonful (half an ounce) in a wineglassful of water three times a day.



After this the patient continued to improve. On 1st December, the thoracic dulness continuing the same, a friction sound was heard over the right base posteriorly; next day the dulness did not reach higher than the upper border of the ninth rib, and friction was heard over the left base also. On 4th December a faint systolic murmur became audible in the mitral area. The patient continued to improve and the dulness over both lungs and heart steadily decreased. On 8th December all medication was stopped, and on 5th January 1871 he was sent to the Convalescent Hospital. After some weeks' rest this patient returned to his work as a tailor, which he still continues. He married a few years subsequent to the illness recorded, and has always had excellent health, in particular he has had no recurrence of his rheumatism. His mitral regurgitation still (1897) persists, but the affection is perfectly mute, though the murmur is loud enough.

Now and then the diagnosis of pericarditis is beset with some difficulties. For example, the sound of friction usually heard at the base of the heart and generally distinct enough, often scrappy and well marked and now and then creaky and obscure, occasionally simulates so closely the blowing sound of a valvular murmur as to be quite indistinguishable from such a murmur by the sound alone. I have most frequently observed this in connection with those latent forms of pericarditis so apt to occur during the course of Bright's disease. There is, however, never any real difficulty in determining between a murmur and a pseudo-murmur of frictional origin. In deciding this matter rhythm is of little consequence, as a pseudo-murmur may be systolic or diastolic, or it may occupy both times continuously, or the reverse—discontinuously and irregularly. And we know that even true valvular murmurs may vary in a similar manner; that is to say, they may be systolic or diastolic, or they may be double, occupying both systole and diastole, or they may be irregular. But a frictional pseudo-murmur has but rarely

the same position of maximum intensity with the murmur which it simulates, the chief exception to this being the pseudo-diastolic aortic murmur. Pseudo-murmurs are strictly localised to the cardiac area, and usually to a very small part of that, not being propagated out of their position of maximum intensity to any extent, and then only equally all round and never in the definite lines of any special valvular murmur. Moreover, all the secondary results of the valve lesion simulated are entirely wanting, and it is only rarely that the natural sounds of the heart are entirely lost in the pseudo-murmur, though they are not infrequently obscured by it. Whenever, therefore, we hear in any part of the cardiac area a sound not distinctly a friction sound but which resembles a murmur, we must always exhaust all the possibilities of valvular origin before we ascribe it to a pericardiac source. The only real difficulty in any such case is when a pseudo-murmur occurs along with pre-existing valvular disease, then indeed it may be impossible to make a perfectly accurate diagnosis, but then too this is of comparatively little importance.

A friction sound heard within the cardiac area is not necessarily of pericardiac origin; it may be pleuritic and yet be audible when the heart alone is acting, the respiration being temporarily suspended. This condition is unusual, and when it does occur an accurate diagnosis is almost impossible. When the friction is entirely absent from the base of the heart over midsternum, and only audible towards the left of the cardiac area, I am disposed to regard it as of pleuritic origin, even though it persists during suspension of the respiration. Undoubtedly this view is a probable one, but it is by no means certainly correct. The subsequent progress of the case may show that the pleura is certainly affected, but that is no proof that the pericardium is not also implicated; or, on the other hand, the pericardium may be assuredly diseased and yet the friction may be wholly due to pleurisy. These problems are among the most difficult we can come

across for a perfectly accurate diagnosis, yet even in the most obscure case there cannot long be any great doubt whether the pleuritic or the pericardiac affection stands alone, or which is the essential disease when the two are combined. Each case must, however, be decided on its own merits; it is impossible to lay down any hard and fast rules applicable to all.

When effusion takes place into the pericardial sac the apex beat is said to be displaced upwards; the true apex is really separated by the effusion from the chest wall. What is now felt is the impulse of the part of the ventricle just above the apex still in contact with the chest wall, and this pseudo-apex appears to rise higher and higher as more and more of the heart gets separated from the anterior chest wall with which the base of the heart always remains in contact. Hence, however great the effusion, basic friction, if it has once been heard, is never effaced. Increased dulness from effusion into the pericardium is first of all to be detected at the base of the heart as an increase of the transverse dulness at the level of the fourth rib; by and by the ordinary pyramidal dulness of the heart, base upwards, is reversed, and we have a pyramidal dulness with the base downwards, while the apex may rise as high as the clavicle or even above it. This dulness may also extend to the left of the usual position of the apex, especially if the patient lies on his left side; but this part of the dulness is mobile, and passes to the right on turning the patient on his right side. Great emphysema of the lung may obscure this dulness, but it never altogether annihilates it. In some cases friction is entirely absent throughout the whole course of the disease, not merely in cases of simple serous effusion, but even when there is an abundance of fibrinous exudation. It is difficult to account for this; a soft gelatinous condition of the fibrine has been alleged as a cause, but it is difficult to believe that differences in this respect are sufficient to account for the entire absence of friction we sometimes have. Feebleness of

the cardiac action is probably at least a contributory cause, and possibly some alteration in the physical condition of the overlying parts, especially of the lungs, may impair their conduction of sound. If with absence of friction we have associated effusion into both pleuræ, then we are forced to surmise the co-existence of pericarditis from general symptoms alone, the most important of these being a rapid, feeble, and dicrotic pulse, with feebleness or absence of the cardiac impulse, and delirium; delirium being even in ordinary rheumatic pericarditis often the only indication of danger, and in all such cases it is a symptom that ought to direct attention to the heart rather than to the head. In January 1868 a case of this nature occurred in Ward VII., in the person of Andrew Dickson, a young man of nineteen, who had double pleurisy with considerable effusion into the left cavity, and somewhat less into the right. He also had occasional hæmoptysis, failure of the cardiac impulse, rapid dicrotic pulse, and considerable delirium; he had no rheumatism and never any pain in the region of the heart. Any pericardiac dulness present was merged in the pleuritic dulness; careful auscultation failed at any time to detect any sound of friction, and though the co-existence of pericarditis was surmised it could never be satisfactorily determined. After death the pericardium was found distended with reddish serum, and both parietal and visceral surfaces coated with shaggy, mammillated, blood-stained lymph. Cases like this are, however, very unusual.

The treatment of pericarditis is almost invariably merged in that of some other disease—rheumatism, Bright's disease, tuberculosis, or pleurisy—and there is no reason why this treatment should be changed because the pericardium becomes accidentally implicated. Stokes has remarked in regard to the so-called active treatment of such cases that “boldness of treatment often betrays the timidity of the practitioner,”<sup>1</sup> and there is no reason why the detection of a little extra

<sup>1</sup> Stokes, *op. cit.* p. 82.



serum or of lymph within the pericardium should lead us to jeopardise our patient by hazardous and unnecessary medication. Bamberger,<sup>1</sup> Niemeyer,<sup>2</sup> Gairdner,<sup>3</sup> and Bennett<sup>4</sup> have all shown that rheumatic pericarditis is a disease which runs its course more favourably the less actively it is interfered with, and that all that is required is fitting constitutional treatment and local palliatives, and that constitutional treatment alone is fitting which is directed to the disease with which the pericarditis is associated, and not to the pericarditis itself. All this is true still, and with modern appliances can be much more readily and efficiently carried out; thus Bamberger and Niemeyer recommend leeching for the relief of pain, but pain can be much more effectually and more rapidly relieved by the hypodermic use of morphine, and, if needful, by the subsequent use of small doses of chloral repeated at regular intervals. Rheumatic pericarditis must be treated simply as part of the general rheumatic attack, with careful attention to keeping the chest warm, to the relief of pain, and to the quieting and steadying of the heart's action.

In occasional instances the question may arise as to whether the pericardium should be tapped or not. It is our duty to obviate death from any preventible cause; if, therefore, the heart's action seems oppressed, and life endangered by the amount of effusion, we ought certainly to tap. In all ordinary cases this procedure is quite uncalled for, and those symptoms that seem to indicate a need for it are only too often signs that the vital powers are so depressed as to render any relief thus obtained merely temporary. In the following case this question of tapping arose, but as the constitutional depression seemed greater than could be accounted for either by the affection of the pericardium or by the amount of

<sup>1</sup> *Lehrbuch der Krankheiten des Herzens* (Wien, 1857), pp. 131-133, etc.

<sup>2</sup> *A Textbook of Practical Medicine* (London, 1871), p. 389.

<sup>3</sup> *Edinburgh Medical Journal*, January 1861, p. 632.

<sup>4</sup> *The Principles and Practice of Medicine* (Edinburgh, 1865), 4th edition, p. 575.

effusion present, the operation was postponed from a feeling that there was something unusual in the case not to be eliminated by tapping. Almost at the last moment my resident, Mr., now Dr. Saundby,<sup>1</sup> did operate, but with only temporary relief, and the nature of the case was such that an earlier operation would have been of no avail.<sup>2</sup>

CASE XXXIV. Thomas Fraser, aged thirteen, was admitted to Ward V., Bed 1, on 21st May 1874, complaining of cough with a feeling of tightness and pain in his chest. His history was that about eight days before his admission he had gone to bed apparently quite well, and in the morning found himself unable to get up from severe illness presenting the symptoms just described. He was a poor ill-thriven boy, whose father had died apparently of phthisis, or at least of some disease accompanied by cough and progressive emaciation; his mother was still alive but sickly. The patient lay easily on his right side; his face was puffy; his insteps slightly œdematous; his countenance expressed both anxiety and pain. His temperature on admission was  $102\cdot4^{\circ}$ , and during the whole of his illness it ranged between  $102^{\circ}$  and  $99^{\circ}$ , being usually a degree higher at night than in the morning. On the day of his death his temperature was  $98^{\circ}$ ; on the previous day it had been  $100^{\circ}$ . His pulse was 80, soft, and very irregular in rate, but specially so in force. He complained of great dyspnoea and tightness across the chest. On inspection the præcordia seemed to bulge more than usual, and upon palpation no cardiac impulse could be perceived. On percussion one inch from the left of the sternum, dulness did not rise above the third rib, but it extended downwards till it merged in the stomach resonance. At the level of the fourth rib transverse dulness commenced two inches to the right of the sternum, and extended to the left across the

<sup>1</sup> Dr. Saundby has published this case with remarks in the *Edinburgh Medical Journal* for March 1875, p. 799.

<sup>2</sup> A full account of this operation, with the history of sixty cases in which it has been performed, will be found in *Paracentesis of the Pericardium*, by J. D. Roberts, M.D., Philadelphia, 1880.

sternum for a distance of five inches more—seven inches in all. On auscultation the heart's sounds were heard normal enough in all the areas when the patient stopped breathing. Over the præcordial region a loud friction sound was audible with the respiration, ceasing when the patient held his breath. The respirations were 36 per minute, and there was great pain on coughing; the expectoration mucous and trifling in quantity. Anteriorly over the lungs the percussion note was normal, except where interfered with by the distended pericardium. Posteriorly there was dulness over the left base. The patient's tongue was slightly coated, appetite defective, bowels loose. The quantity of urine passed could not be estimated, as much of it passed with his stools; what could be collected was of a reddish colour, acid, specific gravity 1027, and contained no sugar, albumin, or bile; chlorides were present, and there was a deposit of urates. The patient's face and feet gradually became more œdematous, but there was no general anasarca of any consequence. Permission was given to tap the pericardium on the symptoms becoming urgent, without much hope of relief from the procedure, but from sheer unwillingness to leave any stone unturned. Accordingly, on the evening of 10th June, the pulse having become imperceptible, Mr. Saundby tapped the pericardium and drew off 30 oz. of pus, with temporary relief. The pulse returned to the wrist—it was 84 per minute—and for a time he rallied, but died at 1.30 on the morning of 11th June. At the autopsy on 12th June some serous fluid was found in the left pleura, and there were recent adhesions over the lower part of the left pleura, chiefly anteriorly. There were 36 oz. of pus in the pericardium, and the heart was firmly adherent to the posterior part of that sac; anteriorly it was covered with purulent lymph. At the base of the right lung there was an abscess close to the pericardium, but no communication could be traced between the two. The rest of the autopsy was unimportant.

In spite of the apparent absence of communication, it

seemed probable that the pus in the pericardium was due to rupture of the abscess of the right lung into the pericardium, the left pleurisy arising from contiguity in a feeble and worn-out constitution. Little relief could be expected from tapping in such a case, but it can be so easily done nowadays by aspiration that it seemed right to give the patient this last chance of having his life prolonged. The intra-pericardiac effusion was never excessive, and the constitutional depression was so great in comparison with the amount of fluid that tapping could only be permitted as a *dernier ressort*, and possibly might as well have been left alone. But it is always right to endeavour to prevent death by every possible means, in the hope that some favourable change may take place. Even in purulent pericarditis this is not impossible, as the elements of pus are more or less present in every pericarditis, and even when the fluid is wholly purulent it may still break up into a pathological cream, become absorbed, and the disease cured, though this can only rarely be the case.

The termination of pericarditis in complete adhesion of its two layers is a matter of great pathological importance, but of little practical interest, because it is almost impossible to diagnose it. We may surmise the existence of this adhesion, but unless in very exceptional cases we can never be certain.<sup>1</sup> Adhesion of the two layers of the pericardium is not necessarily accompanied by any indication of cardiac failure, but where this is present its nature and character depends upon the extent to which the integrity and nutrition of the myocardium is involved. One of the most remarkable instances of this mode of termination of pericarditis is to be found in the Museum of the University of Edinburgh, and is referred to by Dr. Burns in his *Observations on Diseases of the Heart* as one in which the pericardium was unusually adherent, and the ventricles so ossified (they are really atheromatous) that, with the exception of the space of one

<sup>1</sup> *Vide* Lecture I. p. 8.



cubic inch over the apex, they were as "firm as the skull," yet the patient never had any palpitation nor any pain in the region of the heart.<sup>1</sup> Fibrinous effusion into the pericardium may compress the coronary arteries and lead to angina and cardiac dilatation, and an adherent pericardium may even be associated with cardiac hypertrophy, though this must be rare, but it is impossible to predict these alterations, and almost impossible to surmise them even with the history of the case before us.

It has twice occurred to me to find a loud friction sound over the whole cardiac area, without any subjective symptoms whatever. The patients certainly felt themselves not quite right, but there was no rise of temperature, nor any pain; neither was there any other disease present to account for a latent pericarditis, in particular there was no kidney disease. In one of these cases the friction sound came and went in the most inexplicable manner; the variations of intensity in the friction in the other case were evident enough, but were not so remarkable.<sup>2</sup> It is alleged, I believe, by some anatomists that there are muscular fibres in the pericardium, and the phenomena presented by these two cases seemed more easily explicable on the supposition that the heart was periodically and spasmodically grasped by the pericardium, than by any other theory.

Acute endocarditis of a simple character is largely a pathological disease, and not a clinical one. A systolic apex murmur so often supposed to indicate endocarditis, when found in connection with an acute rheumatic attack, is in by far the larger proportion of cases merely an indication of cardiac dilatation of a temporary and perfectly curable character.<sup>3</sup> Any symptoms that may be supposed to be peculiar

<sup>1</sup> *Op. cit.* p. 131.

<sup>2</sup> Robert Hume, admitted to Ward XXXVI., New Royal Infirmary, 23rd June, and discharged 13th July 1880, the friction still occasionally recurring. For some years I saw this man occasionally, looking the picture of health, and to the last the friction persisted without change and equally without detriment to the patient's general health.

<sup>3</sup> *Vide* Lecture VI. p. 167 *passim*.

to this disease, when it does exist, are merged in those of any concomitant pericarditis, or simply in those of the general disease. I have several times had an opportunity of watching the development of stenosis of the mitral valve, from a state of perfect health to the fully-developed pre-systolic murmur, in non-rheumatic cases, but the symptoms exhibited have never been distinctive enough to enable me to lay down any certain rule for their recognition, particularly in the early stages, when recognition would be of most importance. For example, palpitation, simple, rapid action of the heart and arteries, such as we find in pyrexia, not always accompanied by any pain or uneasiness in the cardiac region, is common enough in many forms of disease, and may even exist as an independent neurosis, and yet for many months that may be the only symptom present in a case where mitral stenosis is ultimately developed. At this moment (1898) there is a case under my care, in which, after the lapse of more than a year, there are as yet no definite signs of a stenosis which is surely coming, and even now is faintly shadowed.

Acute ulcerative or septic endocarditis does not seem to be common in our Infirmary, at least we have seen none of it. As its name implies, it owes its virulent and too often fatal character to the presence in the blood of pathogenic organisms. Septic endocarditis is closely allied to pyæmia, and, like its congener, it gives rise to many septic embolisms, accompanied by irregular febrile paroxysms. Unless associated with a valvular lesion, this disease may escape recognition, but the type of the pyrexia, and the characteristic skin eruption, fine reddish spots with a paler centre, serve to exclude typhoid fever and tuberculosis, with which it is apt to be confounded. The disease may be acute, subacute, or chronic; it is not amenable to treatment, but the few chronic cases that have recovered have done so under the internal use of antiseptics with the free administration of stimulants.

Myocarditis belongs to a similar category; it is more often

discovered after death than recognised during life. I shall content myself with giving an account of one or two cases in which more or less extensive myocarditis was found after death, nothing of the kind having been suspected during life.

CASE XXXV. Francis Lynch, admitted first of all on 23rd June 1868, discharged improved on 11th July. Re-admitted on 5th October, discharged improved on 17th December. Again readmitted on 2nd January, and died in Ward VII. on 6th May 1869. This patient had a mitral murmur of a shifting character; at times it was loud and distinct, and again it disappeared entirely for weeks at a time. A tricuspid murmur also present was equally variable. It was impossible to make anything of his cardiac symptoms except that there was a gradual failure of cardiac power, accompanied by an occasional murmur, now distinctly mitral in character, and at other times quite as distinctly tricuspid. He was repeatedly seen by my colleagues, but nothing more definite was elicited. At the autopsy on 6th May 1869 the pericardium was found to contain a considerable quantity of yellowish serum; the heart was enlarged; the apex rounded; the right ventricle projected a little beyond the left. The aortic and pulmonary valves were normal. The circumference of the pulmonary artery measured three inches and a quarter. The tricuspid orifice measured six inches and three-quarters in circumference. The aortic orifice measured three inches, and the mitral orifice five inches in circumference. The right ventricle was dilated and distinctly hypertrophied. The left auricle was somewhat dilated. The left ventricle was in some parts atrophic, but the *musculi papillares* were distinctly hypertrophied. A considerable part of the wall of the left ventricle was atrophied and reduced to half its natural thickness; over this part the endocardium seemed to be continuous. There was no aneurysmal dilatation of the wall, but the cardiac cavity was increased in size by half the thickness of the ventricle. Adherent to the atrophied part of the ventricular wall there was a buff-

coloured clot, firm on its surface, and partially laminated, the superficial layers being redder and apparently more recent. On more careful examination the atrophied part was found to be limited to the inner half of the ventricular wall, the outer half being composed of apparently natural muscular fibre. Quite at the apex, and at the lower part of the septum, the portion atrophied was greater than that remaining, but on tracing it up towards the base, the muscular substance was found gradually to increase until it occupied the whole thickness of the ventricular wall. The fibrous part contained some dense white lines, and was otherwise composed of fine fibrous tissue. On microscopic examination the muscular fibre in every part of the heart was found to be fatty. In the atrophic portion there were found traces of occluded blood-vessels, with granules and crystals of blood-pigment within them. Mingled with these there was a large amount of dense fibrous tissue, with little patches of fat, and traces of muscular fibre. The branches of the coronary artery leading to the atrophied part were occluded. The left pleural cavity contained a considerable quantity of serum, which compressed the lower lobe of the corresponding lung. The right lung was congested and œdematous. There was lateral curvature of the spine in the dorsal region. The rest of the body was not examined.

This patient never had rheumatic fever, and was said to have been perfectly well up to January 1868, when he caught cold, shortly after developed shortness of breath, and ever after continued ailing till he died. It is curiously indicative of the anomalous character of his symptoms that though seen by many physicians, his exact condition was not even surmised.

Another most interesting case is solely represented by the annexed drawings, which exhibit the microscopic appearances found in his myocardium. You see that the connective tissue of the pericardium, the sub-pericardial fat, and the fibres of the myocardium, are all infiltrated with a



hyperplasia of corpuscular elements. The patient to whom this heart belonged laboured under aortic incompetence, but had improved greatly under treatment. He had a good firm apex beat, considered himself quite restored, had got his discharge, and was just about to leave when he died suddenly from asystole. On dissection his heart was flabby, of a yellowish tint, and was pronounced fatty. My resident (the late Professor C. S. Roy, of Cambridge) secured the

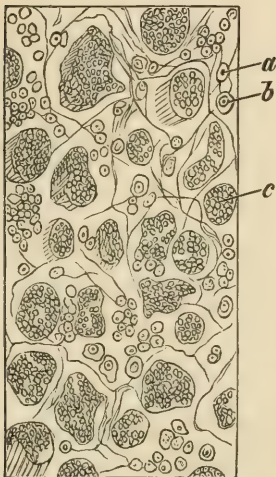


FIG. 29.—Section of muscular tissue of heart. *a*, nucleus; *b*, nucleated cell; *c*, transverse section of muscular fibre.

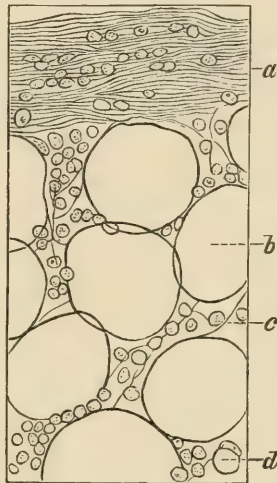


FIG. 30.—Vertical section of pericardium and sub-pericardial connective tissue. *a*, Pericardial fibrous tissue infiltrated with nuclei; *b*, fat cell; *c*, nucleus; *d*, capillary vessel transversely divided.

heart, and, on examining its tissues microscopically, discovered the lesion described. The drawings (Figs. 29 and 30) were made by Dr. Roy, from specimens prepared by himself.

It is impossible to say whether this condition would have ultimately led to suppuration, or to fibrosis of the myocardium.

But perhaps the most remarkable heart of this character that has ever come before me was that of—

CASE XXXVI. Ralph Scott, admitted to Ward V. on

22nd June 1877. This man was forty-five years of age, and of very intemperate habits. He was not known ever to have had rheumatic fever, but had long suffered from cough and shortness of breath, and he was admitted to the Infirmary on account of the severity of these symptoms. He was found to labour under general dropsy with œdematous lungs, a very large heart, and both a systolic mitral and a tricuspid murmur, but nothing peculiar was observed in regard to the action of the heart. Four days after admission, on 26th June 1877, he died suddenly after an epileptiform convulsion.

*Autopsy, 27th June 1877.*—The body was that of a man of rather a large frame, with a good deal of anasarca, especially in the lower extremities. The pericardium was firmly attached over the whole surface of the heart by dense old adhesions. The heart itself was enormously enlarged, weighing 2 lbs.; all its chambers were much dilated, the dilatation of the right ventricle being extreme. The cavity of the left ventricle was greatly dilated, and its wall was much hypertrophied, measuring from three-quarters of an inch to one inch in thickness. The muscular tissue of the upper half of this ventricle was sound, but about the apex the muscular fibre was largely replaced by cicatricial tissue so dense as to creak under the knife. About one inch and a half above the apex there was a pouch-like bulging of the ventricular wall, which would have developed into an aneurysm had life been prolonged. Over the whole area of fibrous induration the pericardial adhesions were specially dense, and the endocardium was thick and of an opaque yellow colour. The aortic orifice was normal in size, measuring three inches in circumference. The aortic valve was competent and its segments natural. The mitral orifice was dilated, admitting five fingers (cone diameter 1·75 inch). The anterior cusp of this valve was natural, but the posterior cusp was in a state of advanced disease; its left half was natural, but the right half was greatly thickened, and was so

curved and bulged out from behind, by the pressure of the blood upon its posterior or ventricular surface, as to press as a rigid, rounded sac against the surface of the anterior cusp. Behind the concave surface of the diseased cusp the posterior wall of the ventricle was hollowed out into a cavity, and presented the same thickening of the endocardium and fibrous induration of the muscular tissue as was noted in regard to the ventricular wall at and near the apex. Owing to the differentiation of the posterior cusp into a sound and a diseased portion, the valve itself looked curiously like a tricuspid rather than a bicuspid valve. The right auriculo-ventricular opening was enlarged, admitting five fingers (cone diameter 1.75 inch). The aorta was normal in size but very atheromatous, and some of its intercostal branches were partially occluded by the consequent thickening of the *tunica intima*, but the coronary arteries were not obstructed.<sup>1</sup> The remainder of the autopsy is omitted as unimportant.

That a heart so crippled could yet discharge its function fairly well for many years, judging from the size of the heart and the fact that its history had been entirely lost, cannot fail to be an encouragement to us in our attempts to remedy even cases that seem apparently hopeless.

I have mentioned that hypertrophy of the heart may be a result of pericarditis quite apart from valvular lesion. This does happen occasionally, just as we also have apparent hypertrophy of the heart from the development of gummata within the myocardium, yielding to anti-syphilitic remedies, but what so often passes under the name of idiopathic enlargement of the heart is simply the physiological result of those tissue changes that occur in our progressive development. When after middle life distressing symptoms attract attention to the heart, in by far the larger proportion of cases these will be found to depend upon some disturbance of the nutrition or of the innervation of the myocardium interfering with and modifying this normal senile enlargement of the

<sup>1</sup> Condensed from the *Pathological Reports of the Royal Infirmary*.

heart. Many of the most interesting, and some of the most widespread varieties of cardiac disease are to be found in this connection, but they depend primarily upon arterial development and have been treated of elsewhere.<sup>1</sup>

<sup>1</sup> Vide *The Senile Heart, its Symptoms, Sequelæ, and Treatment*. London : A. and C. Black, 1894.



## LECTURE XIV

### ON THE THERAPEUTICS OF CARDIAC DISEASE

IN speaking of the various forms of cardiac disease I have already alluded, somewhat cursorily, to the method of treating them. At present I intend to go a little more fully into cardiac therapeutics generally, the remedies commonly used, and the indications for their employment.

First, let me point out that as a preliminary to the treatment of any disease, it is needful to make an accurate diagnosis of the general condition of the patient, as well as of the state of the organ chiefly at fault. The prominent symptom complained of affords an important clue to the nature of the case, as well as a guide to the treatment. But that any symptom may be useful in this way we must clearly understand what the patient means by his complaint, as well as what the symptom means to us. In heart affections, for instance, palpitation is a common complaint, and to an ordinary patient it means anything abnormal in the rate or rhythm of the heart's action. But palpitation, thus understood, is a symptom having various pathological meanings, and each of these indicates an entirely different line of treatment (*vide* p. 276). Again, dropsy is a symptom readily noticed and commonly enough associated with a diseased heart, but the association may be quite accidental, and to accept concomitance as an indication of relative connection might result in sacrificing to routine a life that a more intelligent differentiation might save. Breathlessness is another symptom often connected with cardiac failure, but

this also frequently depends upon causes wholly unconnected with the heart, and demanding diverse modes of treatment.

We must also remember that if a patient comes complaining, it is our duty to relieve his sufferings, but if we accidentally discover a cardiac lesion or murmur in any one apparently healthy, there is not usually any call to make such a one an invalid, or to employ any special treatment. The chief exception to this is any young spanæmic person, girl or boy, with a so-called functional murmur, as the hearts of all such patients must be attended to whether they complain or not, otherwise they may find themselves seriously handicapped later in life.

In estimating the value of any treatment it is an unwise error to suppose that recovery or improvement has been necessarily due to the use of any special drug. In past ages many cardiac patients recovered from ruptured compensation without any material aid from drugs. And in the present day many recover, even after dropsy has set in, under the influence of diet and rest alone, and this enables us to understand the recoveries that often take place under the use of drugs of no great activity.<sup>1</sup> Powerful drugs are always potent for evil as well as for good, and in the hands of the less skilful a less active drug may be actually more useful than one which is more energetic, with the management of which the practitioner is less acquainted.

In the treatment of cardiac disease it is, before all things, needful to keep the patient cheerful and free from anxiety. If there is cause for apprehension,—and no heart case is altogether free from this,—it is well that the friends should be warned; but if these are injudicious, it is better to run some risk of being misunderstood rather than disturb the equanimity of the invalid.

With these few preliminary remarks I pass now to the consideration of those remedies chiefly employed in the

<sup>1</sup> Vide *Transactions of the Medico-Chirurgical Society of Edinburgh* (Session 1895-96), vol. xv. (new series), p. 142.

treatment of heart affections; they are not very numerous, but they are very valuable, and considering the importance of the organ upon which their energy is chiefly expended, every practitioner ought to make himself thoroughly acquainted with the action of each of them.

Dotted all over our upland pastures there is no nobler plant of our indigenous flora than the *Digitalis purpurea*, and there is no more potent benefactor to mankind among the many constituents of our *materia medica*. For over a hundred years digitalis has been known as a sovereign remedy for dropsy, but it is chiefly within the last twenty years that its true action has begun to be recognised. Even yet the profession are hampered by the idea that digitalis is a dangerous sedative to the heart—an idea that dates back to the time not so long past, when digitalis was supposed to possess a number of distinct actions, all more or less antagonistic to one another.<sup>1</sup> Now we know that the fundamental action of all that group of plants to which digitalis belongs, to which it gives its name, and of which it is *facile princeps*, is to increase the elasticity of muscular fibre so that it expands more slowly and contracts more perfectly.<sup>2</sup> The effect of this upon a hollow muscle like the heart is that it dilates more slowly and empties itself more completely; residual accumulation is thus prevented, and any dilating influences are counteracted. As all the blood passes many times through the heart for once that it passes through any other muscle,<sup>3</sup> the myocardium is powerfully affected while the other muscles still remain practically uninfluenced. The muscles of the arterioles are also early and powerfully affected, though not to the same extent as the heart, as only the blood

<sup>1</sup> Christison's *Dispensatory* (Edinburgh, 1842), p. 400. Also *Edinburgh Medical Journal* (February 1870), p. 743.

<sup>2</sup> Schmiedeberg, "Beiträge zur Kenntniss der pharmakologischen Gruppe des Digitalins," *Archiv für experimentelle Pathologie und Pharmacologie*, Bd. xvi. S. 149.

<sup>3</sup> And in this respect we must not forget that "the circulation through the myocardium may be partly carried on by the *foramina Thebesii*."—*British Medical Journal* (2nd October 1897), p. 882.

going to the district supplied by these vessels passes through them.<sup>1</sup> The increased elasticity imparted to the myocardium by digitalis enables it to expand and contract more perfectly, and so to throw a larger blood-wave into the arteries, and as from the increased elasticity of the muscles of the arterioles<sup>2</sup> the arteries empty themselves more slowly, the blood accumulates within them, the blood-pressure gradually rises, and in accordance with Marey's law the heart's action is slowed.<sup>3</sup> No doubt this slowing of the heart is partly due to the action on the cardio-inhibitory centre, but the action on the muscles is quite sufficient to account for all the phenomena observed, and it is with it we are chiefly concerned. The result of this rise in the blood-pressure is that the secretions are improved, and all the tissues of the body, amongst them the myocardium, are fed with richer blood at a higher pressure, so that metabolism is more perfect and every function more efficiently performed. As the blood accumulates within the arteries the veins are correspondingly emptied; the serous soakage of the tissues is reabsorbed, and the excess of water that thus gets into the blood is removed by the kidneys, so that for a time the urine is increased.

The improved metabolism of the myocardium enables it to discharge its function more perfectly, and to resist all those deteriorating influences to which a weak heart succumbs, any hindrance to the circulation is fully compensated, and despite the existence of a valvular lesion, the individual may descend into the vale of years wholly unconscious of the possession of a heart.

All the benefits we obtain from digitalis are inseparably connected with its tonic action; they flow from the power

<sup>1</sup> Stockman, *New Official Remedies* (London, 1891), p. 58.

<sup>2</sup> This action on the arterioles has been anew proved experimentally by Dr. Lauder Brunton and Tunnicliffe, *Journal of Physiology*, vol. xx. p. 354.

<sup>3</sup> *Physiologie Médicale de la Circulation du Sang*, par le Dr. E. J. Marey (Paris, 1863):—"Le cœur bat d'autant plus fréquemment qu'il éprouve moins de peine à se vider."



that digitalis has of increasing muscular elasticity, and the improved metabolism of all the tissues, but specially of the myocardium, that follows this. Digitalis is no opium to the heart, as it has been called, it does not relieve by narcotising, it soothes cardiac irritability and assuages some forms of cardiac pain by improving cardiac metabolism, failure of which has been the cause of both irritability and pain. These benefits are readily obtained by moderate doses of the drug, and though great advantage sometimes accrues from the judicious employment of larger doses, yet the frequent repetition and long continuance of even small doses are often sufficient to secure the very best results. The abuse of the drug, so frequently followed by distressing if not alarming symptoms, proceeds upon an entire misconception of the true action of digitalis.

Digitalis in every form is absorbed with difficulty and only slowly excreted, hence if even a moderate dose is repeated at too short an interval the drug accumulates within the system, and ere long symptoms of intoxication appear. This faculty of accumulation digitalis shares with many other drugs. Unfortunately for its own reputation, its sphere of usefulness lies among a class of cases in which sudden death is no uncommon occurrence, and many a death has been unjustly ascribed to this cumulative action quite irrespective of the dose administered. Even yet there is an idea of danger connected with this drug which limits the usefulness of this invaluable remedy, and favours the adoption of less active and sometimes of even more dangerous drugs. But deaths from digitalis among those otherwise healthy are amongst the rarest of occurrences; only a very few are on record. A full account of one has been published, in which 211 grains of the powdered leaves were taken within five weeks before the fatal result.<sup>1</sup> Sickness, diarrhoea, faintness, and slow pulse had prevailed during fully four of these weeks. With ordinary medicinal doses we need never apprehend any

<sup>1</sup> Köhnhorn, *Vierteljahrsschrift für gerichtliche Medizin* (1876), S. 278.

danger, and we are certain of plenty of warning of even the approach of discomfort.

As may be readily supposed, saturation is more easily brought about in stunted and anæmic individuals than in those who are more bulky and plethoric. But any risk from an overdose must be very slight indeed, as in all my experience of many years I have never seen any unpleasant result follow a somewhat free use of digitalis. A tonic dose fitted to improve the nutrition and increase the energy of a feeble heart is always a moderate one, and must only be repeated after a sufficient interval. One grain of the powdered leaves, or an equivalent dose of any of the other preparations of the drug, given every twelve hours, will be found to be quite an efficient dose for this purpose. The feeble heart steadily improves, and after a time an imperceptible impulse becomes perceptible, and a feeble impulse a strong one, and the patient recovers a sense of wellbeing and a capacity for exertion to which he has long been a stranger. A dose such as that described, and less will rarely do, may be continued for years, if necessary, with nothing but increasing benefit to the patient, and without the slightest need for any anxiety as to the possible occurrence of any disturbing saturation. The dose given may even be smaller than one grain, and it may be given oftener than every twelve hours, but in that case the various doses may not amount to more than one grain of the drug within each twelve hours without risk of symptoms of saturation appearing.

A feeble heart beats more quickly and less forcibly than in health, the ventricles empty themselves less completely, there is residual accumulation within their cavities, the arteries get depleted and the veins over-filled. The result of this venous stasis is soakage of all the tissues, and it is the removal of this soakage and the improvement of the general metabolism that imparts the sensation of wellbeing that follows the tonic use of digitalis. By persistence of all the abnormal conditions this soakage ultimately collects as dropsy

in the more dependent parts of the body and within its cavities. This dropsical accumulation is chiefly of consequence as an indication of heart failure, but in the tissue interspaces, especially in the extremities, it also constitutes an important peripheral obstruction to the circulation, increasing the work of an already failing heart.

In cases of dropsy our forefathers employed digitalis empirically as a diuretic, often with what seems to their more timorous successors to be a dangerous freedom. They gave 10 to 20 grains of the leaves in powder or infusion every hour or every two hours till nausea or sickness occurred; the drug was then stopped, and this acute poisoning does not ever seem to have been followed by untoward results, the diminution or suppression of urine which accompanied it was, in two or three days, followed by copious diuresis, which removed the dropsy. Nowadays we do not employ digitalis empirically as a diuretic, we use it as a tonic to the myocardium, and we find that tonic doses suffice to remove soakage and even a moderate amount of dropsy. But when the heart is considerably dilated larger doses may be given with advantage to restore the normal elasticity of the myocardium and to overcome the tendency to dilate. Still for this but a moderate dose is necessary; 3 grains every eight hours will be found very efficient, and the interval will probably soon require to be increased to every twelve hours, or the dose diminished. The larger the dose and the shorter the interval the more watchful we must be for symptoms of saturation. These symptoms—which may be looked for so soon as 30 or 40 grains have been ingested in larger doses than one grain, and at a shorter interval than twelve hours—are diminution of a primary diuresis, abnormal slowing of the pulse, or nausea, more rarely diarrhœa. If the medicine is stopped on the first occurrence of one or other of these symptoms the patient will suffer no damage, nor will the physician be ever disappointed in any reasonable expectation. Such is Withering's statement, and with it I entirely agree.

When dropsy is present and saturation occurs the primary diuresis diminishes, and almost complete suppression may last for two or three days ; diuresis then recurs and may continue till all the fluid is drained off, or it may be kept up by moderate doses at regular intervals of eight or twelve hours. Apart from any diuretic action, these cumulative doses, by improving the tone and elasticity of the myocardium, not only enable it to resist dilating influences, but in favourable circumstances are able to restore a dilated heart to its normal dimensions.

Much larger doses than those indicated have been therapeutically employed without detriment. It is on record that a young woman affected with mitral stenosis took for six years  $4\frac{1}{2}$  grains of digitalis in infusion night and morning, with nothing but continuous benefit. Without her medicine she was unable to work, her limbs were oedematous, flashes of light dazzled her eyes, a rushing sound disturbed her hearing, her heart felt full to bursting, and her urine was suppressed. All these symptoms disappeared within a few hours after her medicine was repeated, and she was once more an active woman.<sup>1</sup> But such a case must be exceptional, and so large a dose cannot be recommended.

The first effect of a moderate but cumulative dose of digitalis is slight slowing of the pulse, with increase of its tension, and a gradually increasing flow of urine. After the ingestion of about 30 grains in cumulative doses, we look out for signs of saturation. With moderate doses at medium intervals these are an abnormal slowing of the pulse, and a diminution of the flow of urine. Should these indications be neglected they are speedily followed by cardiac irregularity, the slow heart beat quickens, falters, and intermits on the slightest exertion. Unless we remember that after 30 grains of digitalis have been ingested in cumulative doses the point of saturation is approached and are watchful, a slight drop in the pulse-rate or in the quantity of urine is apt to pass un-

<sup>1</sup> Bälz, *Archiv der Heilkunde* (1876), S. 468.



noticed, and the patient may get plunged into all the discomforts incident to vagus paralysis, while we are scarcely aware that the drug has begun to affect him. This, however, is not the fault of the drug, but the result of inattention on the part of the practitioner, and can only occur when cumulative doses are given.

The abnormally slow pulse of digitalis saturation seems quite devoid of danger, but the rapid irregular pulse of digitalis poisoning (vagus inhibition) indicates a condition not devoid of risk, but one which may be safely tided over by omission of the drug, strict adherence to the recumbent posture, and the moderate use of stimulants.

The primary action of digitalis is thus that of a tonic to the heart, it increases the elasticity and improves the nutrition of the myocardium, refills the arteries, depletes the veins, and removes dropsy. All this can be done by moderate doses, quite incapable of producing any discomfort. But when the heart is much dilated, or when the dilating forces act powerfully—as in aortic regurgitation,—to secure rapid action larger doses must be employed, and then comes the need for watchfulness lest saturation and its unpleasant though not necessarily dangerous symptoms should steal upon us unawares. From the nature of its action digitalis may be beneficially employed in all forms of cardiac failure, whatever be the character of the valvular lesion with which it may be connected. Corrigan long ago sounded a note of warning in regard to the use of digitalis in aortic incompetence; he looked upon that drug as a pure sedative, and regarded the prolongation of the diastole as a source of increased danger to the heart. It is a very instructive proof of how ill understood the true action of digitalis is even yet that a similar idea is still held by some. Nay, more, the prevalence of such an idea is also a proof of how theory has been allowed to usurp the place of observation, because the most cursory observation is sufficient to satisfy us that undue prolongation of the heart's diastole is never requisite to enable

us to obtain all the benefit we desire. In aortic incompetence, as in all forms of cardiac failure, the heart's action is quicker than usual, Corrigan reckons it as averaging 110 per minute,<sup>1</sup> and it is neither needful nor desirable to slow it down below the normal (say to 70), and he would be a bold man who should affirm that a restoration of the heart-beat to its normal rate is in the least likely to be in any way injurious (*vide antea*, p. 105).

Digitalis fails occasionally; thus it has no action in the often protracted and ingravescient asystole of a moribund heart. At first we may fail to recognise this condition as terminal, and we are bound to employ that remedy which seems to hold out the most hope of advantage, but when the conditions are fully recognised we have no right to be disappointed at any failure of result.

Since the days of Withering it has been known that when "the limbs in anasarca are solid and resisting we have but little to hope" from digitalis. The analogy here is with those senile hearts whose dilatation is due to loss of arterial elasticity; in both cases digitalis is of no use and may even be hurtful until the peripheral obstruction is removed. In the one case the peripheral obstruction is due to pressure of fluid on the arterioles and is readily removed by purgation or drainage. In the other the obstruction to the circulation caused by loss of arterial elasticity is aggravated by the digitalis action on the arterioles, and this is counteracted by the combination of a vascular stimulant with the cardiac stimulant.<sup>2</sup> In both cases digitalis acts like a charm when delivered from the incubus of peripheral obstruction.

In dilatation of the right ventricle following considerable stenosis of the mitral opening, or originating in limitation of the area of the pulmonary capillaries due to destruction of the walls of the air-sacs by emphysema, digitalis acts as a tonic and improves the condition of the heart, but it can

<sup>1</sup> *Edinburgh Medical and Surgical Journal* (April 1832), p. 241.

<sup>2</sup> *The Senile Heart*, pp. 266 and 274.

never induce contraction of the right ventricle because of the permanent and incurable nature of the cause of the dilatation.

Lastly, digitalis is said to act badly and even to be positively injurious in cases of fatty heart. But hearts diagnosed as fatty are, as a rule, only feeble and dilated, and it would be a sad mistake to deprive such hearts of the benefit they are so certain to receive from digitalis from the dread of an unrecognisable chimæra.<sup>1</sup> The following case affords an apt illustration of the impossibility of diagnosing a fatty heart, as well as of the benefit that feeble hearts, even though fatty, sometimes derive from digitalis.

CASE XXXVII. John Steven, a sailor, aged sixty-four, admitted to Ward V. (from the Clinical Wards) on 24th July 1875, complaining of cough, shortness of breath, palpitation, brawny swelling of the legs, and some ascites. He admitted having been a hard drinker. The cardiac action was rapid, irregular, and intermittent. The apex beat was quite distinct though not forcible, and was felt outside of and below its normal position. The sounds at the base of the heart were obscured by bronchitic rhonchi, but no murmur could be distinguished. The first sound in the mitral area was impure but without murmur; a murmur of tricuspid regurgitation was, however, occasionally to be heard. The patient's liver was not enlarged, but he had a trace of albumin in his urine, which three weeks before his death became considerably increased. His sputa were rusty for a week before his death. This patient was regarded as suffering from a weak, dilated, and probably fatty heart; the latter part of the diagnosis being not based upon any physical sign, or upon any symptom, but mainly upon the patient's previous drunken habits.

<sup>1</sup> "Die einfache Erfahrung, dass man in vielen Fällen von Herzdilatationen mit starker Unregelmässigkeit des Pulses bei der Section oft nur ein geringe oder gar keine Fettmetamorphose findet, während schwere Verfettung der Muskelatur ohne alle Symptome von Seiten des Herzens verlaufen können, die Erfahrung also dass die Muskelverfettung nicht in directen Verhältniss zur Schwere der klinischen Symptome steht, zwingt uns ein besonderes Krankheitsbild 'Fettherz' aufzugeben."—Fraentzel, *Die idiopathische Herzvergrösserung* (Berlin, 1889), S. 191.

During the ten weeks he was under treatment in Ward V. the only relief he obtained was from large doses of digitalis; 15 minims of the tincture every four hours was his average dose, sometimes he got more, never less. I did not see him for two weeks before his death, and at the time I left there still seemed a chance of the digitalis restoring his cardiac energy; he died, however, on 5th October 1875, apparently from asthenia, culminating in asystole (ingravescent asystole). On dissection, on 7th October, the body was found to be greatly anasarcaous and considerably decomposed. The heart weighed 24 oz., the parietal and visceral layers of the pericardium were adherent throughout; the connecting lymph was recent, except posteriorly, where it was somewhat fibrous. The heart was both dilated and hypertrophied; all its valves were thickened by atheroma but competent, except the aortic valve, which was slightly incompetent. The cavities of the heart were filled with black fluid blood. The cardiac muscle was everywhere soft, friable, and easily penetrated by the finger. On microscopic examination it was found to be in an advanced state of fatty degeneration, the striation being entirely lost. The lungs were firmly adherent over their whole extent, intensely congested, and of a dark colour; the right weighed 5 lb. 3 oz., the left 2 lb. 4 oz. The liver was fatty, congested, and weighed 3 lb. 13 oz. The spleen was congested, and its capsule thickened; it weighed 5 $\frac{3}{4}$  oz. The kidneys were in an advanced state of fatty degeneration, as proved by microscopic examination; the right weighed 8 oz., the left 7 oz.<sup>1</sup> To those who had observed the great benefit this patient derived from the use of digitalis during his lifetime, and who afterwards saw the extremely fatty state of his heart, nothing could more instructively show both the power of the drug over any trace of muscle that remained, and also the small amount of danger to which fatty hearts are exposed by the administration of digitalis. Even in aged people with a feeble impulse and a tendency to syncope, in whom fatty

<sup>1</sup> Extracted from the *Pathological Records of the Royal Infirmary*.



degeneration may reasonably enough be suspected, I have never seen any reason to withhold digitalis, and have hitherto been rewarded for my boldness.<sup>1</sup>

Digitalis contains three important principles, digitalin, digitalein, and digitoxin. Of these, digitoxin is absolutely insoluble in water, but soluble in alcohol, and more or less crystallisable. It is probably the most active ingredient of the crude drug; and though regarded by some as less reliable as a diuretic, and more prone to excite nausea, these results are more probably due to its energy rather than to any specific properties it possesses. From their tardy absorption the active principles of digitalis are prone to excite local inflammation when employed hypodermically. Fortunately rapid action is not of much consequence in those cases in which most benefit is to be expected from digitalis; when rapid stimulation is of importance, we have other drugs to employ. Where the dose of the crude drug is so small as that of digitalis is, it is scarcely worth while employing active principles, and it seems sufficient to make ourselves acquainted with the crude drug and with its galenical preparations. Any inquiry into the most suitable preparations of digitalis and their most appropriate doses must be conducted with due regard to the action we wish to secure, and perhaps a little to our geographical position, a somewhat paradoxical statement, yet latitude appears to have some influence on the potency of this drug. The powder of digitalis, prepared from carefully dried leaves not more than one year old, was regarded by Withering as the most active preparation of the drug, and he considered that 30 grains of the powdered leaves in bulk were about equal to 40 grains in infusion, and that this quantity in divided doses could generally be taken before the occurrence of nausea. Since the days of Blackall,<sup>2</sup> however, the powdered leaves and the infusion have been regarded as of nearly identical

<sup>1</sup> Vide *The Senile Heart*, p. 216.

<sup>2</sup> *Observations on the Nature and Cure of Dropsy* (London, 1814), p. 312.

value, and the action of any preparation as reasonably calculable from the number of grains of powdered leaves it represents. One grain of the crude drug, or its equivalent in one or other preparation, given every twelve hours, is a purely tonic dose in all but the most exceptional cases. In these exceptional cases even this dose, and in all others any larger dose or the same dose repeated at shorter intervals than twelve hours is cumulative in its action. This is indicated, after the ingestion of about 30 grains, by an abnormal lowering of the pulse-rate and a reduction in the flow of urine. The infusion ranks next to the leaves in usefulness and probably in efficiency, and it lends itself readily to combinations with vascular stimulants, which are so indispensable in certain forms of cardiac disease. Half an ounce of the British Pharmacopœia infusion contains rather more than one grain of the leaves, but may be accepted as the equivalent of that quantity. The tincture contains about a grain in each 10 minims, and that therefore is the tonic dose of this preparation. The tincture is a very uniform preparation, acts well and combines readily with other tonics, but does not combine so well with vascular stimulants. Merck's digitalin, probably mainly digitoxin, is a reliable and convenient preparation, as is also Nativelle's, said by Brunton to be also mainly digitoxin; each of Nativelle's granules contains one-quarter of a milligramme (0.003858 of a grain), and they are most convenient and reliable when employed as a cardiac tonic in doses of one granule once or at the most twice a day. In larger doses or when given at shorter intervals both Merck's and Nativelle's preparations are prone to excite nausea, it is not therefore convenient to employ either of them for any other purpose than as a tonic to the heart. Purging is reckoned by Withering to be one of the actions of digitalis; it occasionally occurs, but whether *propter* or merely *post* I have never been able to satisfy myself; when purging does occur diuresis is always lessened but not necessarily put a stop to.

We can, by gradually increasing the dose of digitalis, so contract a dilated heart as to reduce its sounds to the toneless tic-tac of an embryonic heart, but there is no object to be gained by this, as this condition cannot be long maintained, and the heart speedily redilates when the drug is omitted. Moreover, there is probably no advantage to be gained by enforced contraction of the heart; our main object is to restore the elasticity and improve the energy of the myocardium, and when this is done the heart is either restored pretty much to its former condition, or any remaining dilatation is fully compensated, and remains indefinitely innocuous.

In using large doses of digitalis carelessly the primary slight diminution of the urinary flux and the oftentimes but short-lived abnormal slowing of the pulse may be unmarked, and the thumping pulse and heart-beat of commencing saturation may pass unnoticed into the arrhythmic pulse of digitalis poisoning, a state that may, however, be soon remedied by omitting the drug, keeping the patient in bed, and giving stimulants moderately. At times instead of irregularity we have a *pulsus bigeminus* developed (*vide antea*, p. 274), but this seldom happens unless digitalis has been given for some considerable time, and it occasionally follows the use of a mere tonic dose where that has been continued for a year or two. No risk seems to attend this condition, and it passes off in no long time when the drug is omitted.

In digitalis we have a remedy of undoubted activity and of remarkable utility in all cases of heart complaint, but like every other powerful agent it has certain disadvantages, and, carelessly employed, it may give rise to symptoms which are always unpleasant, if but rarely dangerous. But inasmuch as we can obtain from digitalis results that are not only valuable but also permanent, and not to be gained by the administration of any other drug, it is certainly worth while to make ourselves thoroughly acquainted with the use of so important a drug, which has the additional advantage of being indigenous. Even when employing tonic doses it is better to

have the patient under observation for a week or two before finally fixing on the appropriate dose. Should there be any marked idiosyncrasy, which is quite unusual, if digitalis is given for other than merely tonic purposes it is better to give full doses for a short period rather than smaller doses for a longer time. But whatever the dose employed the desired results will never be observed till a certain amount of saturation has been induced.

If we take one grain every twelve or every twenty-four hours as a tonic dose, then from 3 to 7 grains every four hours are the extremes between which a cumulative dose may oscillate, and after the ingestion of from 30 to 45 grains, that is, after fifteen doses in the one case and nine in the other, nausea, abnormal slowing of the pulse, or diminution of the flow of urine may be expected to occur, if they have not been previously observed. From one to three days subsequently, in cases of dropsy, the full diuretic effect of the drug may be expected, the flow of urine will rapidly rise to about 200 ounces in the day, and in favourable cases will continue to flow till all the cavities are emptied. Occasionally the diuresis requires to be maintained by the administration, at regular intervals, of smaller doses. The delay of diuresis does not seem to depend upon any contraction of the arterioles, at all events it has not been terminated or in any way affected by repeated inhalations of nitrite of amyl. It is of consequence to remember this delay in the diuresis that follows the ingestion of full doses of digitalis, as we are otherwise liable to ascribe this belated flow to the influence of some comparatively inert drug that has been administered during the interval.

The fundamental action of digitalis furnishes a sufficient answer to the question as to the form of heart affection in which we are to employ it. In valvular lesions in the stage of compensation, which we know is never complete, digitalis is never contra-indicated, but only tonic doses are applicable, and in this dose the drug will do nothing but good, however long it may be continued. When compensation is ruptured



—*la période asystolique*—digitalis is imperatively required in every form of heart affection, it must be given more or less freely according to the symptoms, and generally in cumulative doses. As such doses lead sooner or later to saturation they must be watched, and the drug omitted or the dose reduced when symptoms of this appear. In spanæmic hearts digitalis is often required ; generally tonic doses suffice, but when there is considerable dilatation cumulative doses may be advantageous, though they are not always successful in contracting the heart so perfectly as we would like. A neurotic heart is always weak, and tonic doses of digitalis are always useful ; after middle life such a heart is almost invariably gouty (senile), and digitalis is not well borne unless conjoined with some vascular stimulant.

The only other member of the digitalis group that has succeeded in obtaining special recognition from practitioners is *Strophanthus Hispidus*, an African plant belonging, not to the *Scrophulariaceæ*, but to the *Apocynaceæ*. In its native habitat it is used as an arrow poison, and it has been introduced to our *materia medica* as a remedy in heart affections mainly on the recommendation of Professor Fraser. Though strophanthus belongs to the same group as digitalis, it acts very dissimilarly. Digitalis acts equally on all muscular fibre, and only more powerfully on the heart and muscles of the arterioles because these organs, owing to the conditions of the circulation, receive from the blood a larger dose of the drug within a given time than the other muscles (*vide antea*, p. 359). The conditions as regards the circulation are permanently similar, and if strophanthus acted on all muscular fibre after the manner of digitalis, there would be a similar ratio between the action of the two drugs on the heart and on the arterioles, but this is not the case. Strophanthus acts *three thousand times more powerfully* on the heart<sup>1</sup> than digitalis, but it acts *one hundred times less*

<sup>1</sup> Fraser on "*Strophanthus Hispidus*," *Transactions of the Royal Society of Edinburgh*, vol. xxxvi. pt. 2, p. 403.

*powerfully* than digitalis upon the muscles of the arterioles.<sup>1</sup> There is thus no similarity whatever between the action of the two drugs. From the entire absence of any appreciable action by strophanthus on the arterioles, the blood flows freely from the arteries into the veins, and any rise of blood-pressure that may occur is due entirely to the ventricular systole. The typical (therapeutically induced) strophanthus heart has a prolonged diastole, during which the arteries have a longer time than usual to empty themselves, and as the ventricle has been filling itself during this period, its systole sends an unusually large blood-wave into unusually empty arteries—precisely what we have in bradycardia. The momentary rise of blood-pressure passes rapidly with the blood-wave down the unfilled arteries; it does not, therefore, persist so long as the ventricular systole, and is consequently of shorter duration than even the third of a cardiac cycle. So evanescent a rise in blood-pressure can, however, have no appreciable effect either on general metabolism or upon that of the myocardium. Herein the action of strophanthus differs essentially from that of digitalis; there is no tonic action, no improvement of the cardiac energy,<sup>2</sup> and the powerfully stimulating action of strophanthus on the heart itself must tend to exhaust an already feeble myocardium.

Moreover, the action of strophanthus on the heart is two-fold;<sup>3</sup> in small doses it may arrest the heart in diastole, and in large doses it may force it into a fatal systole.

Like all its congeners of the *Apocynaceæ* strophanthus is a cardiac poison, and not a cardiac tonic. It forces the heart into increased energy of movement without providing for any corresponding improvement in its metabolism, hence the heart must draw upon its reserve, and the patient is only

<sup>1</sup> *Ibid.* vol. xxxvi. pt. 2, pp. 438, 453.

<sup>2</sup> "The results derived from strophanthus are certainly not so lasting as those which follow the use of digitalis."—Binz, *Lectures on Pharmacology*, New Syd. Soc. edition, vol. i. p. 259.

<sup>3</sup> Fraser on "Strophanthus Hispidus," *Transactions of the Royal Society of Edinburgh*, vol. xxxvi. pt. 2, p. 401.

saved from dire disaster by the benefit he derives from rest, warmth, and nutritious food,—that is, by the improvement in his environment generally. It has been claimed that the absence of action on the arterioles is to be reckoned to the advantage of strophanthus in those numerous cases where vascular stimulants require to be conjoined with digitalis to modify its action in raising the blood-pressure. But these cases are precisely of that class in which before all others it is of importance to improve cardiac metabolism, and to employ in such cases a drug like strophanthus, which stimulates and exhausts the energy of the heart without improving its metabolism, must be a distinct menace to the integrity of the organism. Strophanthus is thus uncertain in its dose, and may be dangerous in its action, and though not cumulative it may be injurious or even fatal without warning, so that from this point of view there may be worse things than the early indications of saturation which digitalis gives us in those symptoms we are accustomed to ascribe to accumulation.<sup>1</sup>

*Nux vomica* is an excellent cardiac tonic, but as its utility depends upon its active principle, it is more advantageous and conduces to greater accuracy of dosage to employ the *liquor strychninæ hydrochloratis* rather than any of the more crude preparations. Strychnine has a special stimulating action on the nervous system generally, hence it stimulates and renders more excitable the vaso-motor centre and the cardiac ganglia, probably even energising that primordial power of spontaneous movement possessed by the cardiac muscular fibre itself.<sup>2</sup> In virtue

<sup>1</sup> For fuller information as to the action of strophanthus I may refer to Professor Fraser's papers on "Strophanthus Hispidus," in the *Transactions of the Royal Society of Edinburgh*, vol. xxxv. pt. 4, and vol. xxxvi. pt. 2; also to a paper by myself in the *Transactions of the Edinburgh Medico-Chirurgical Society* (new series), vol. xv. p. 141, entitled "A few more Words on Strophanthus."

<sup>2</sup> An interesting account of the action of strychnine on the heart will be found in a paper by Dr. Lauder Brunton and Professor Cash at p. 229 of vol. xvi. of *St. Bartholomew's Hospital Reports*.

of this action on the heart and nerve centres strychnine increases the heart's force and raises the blood-pressure, acting very much like digitalis but more on the nerve centres and less upon the muscles themselves. Moreover, strychnine is an admirable tonic to the stomach, particularly in those catarrhal conditions accompanied with venous congestion so commonly present when the circulation is feeble. In this way digestion is improved and the blood enriched, so that every organ in the body, and the heart in particular, gets better nourished and better fitted for the discharge of its function. Strychnine is cumulative in its action, so that we require to be both accurate in our dosage and regular in the times of administration, but with strict attention to these matters it may be given continuously for many years with nothing but increasing benefit. The proper dose of the *liquor strychninæ* is 5 minims ( $\frac{1}{26}$  of a grain of strychnine) every twelve hours, this for the larger number of mankind is a perfectly safe dose; now and then idiosyncrasy turns up, and for it we must be prepared. In anæmic patients, for example, there is occasionally an intolerance of strychnine, and if employed at all it must be given in almost infinitesimal doses. Only rarely can more than 5 minims of the *liquor* be given in the twelve hours, and 15 minims instead of 10 in the twenty-four hours are apt to be followed in no long time by symptoms of saturation. In cases of feeble hearts strychnine alone often suffices, but if there is marked dilatation digitalis should always be combined with it. From its action on the gastric mucous membrane strychnine is specially useful in cases of feeble hearts whose action is made irregular by gastric reflexes, and in such cases it is of advantage to combine it with arsenic, and sometimes with iron. In these cases a few weeks often work wonders, but in most we may have to wait for months before we reap the full benefit.

*Arsenic* is also one of our most valuable remedies in the treatment of heart disease. Its effect in relieving the pain



of angina is occasionally marvellous; it acts most beneficially in those congestive conditions of stomach that so often accompany a feeble circulation; add to this that its tonic influence on the lungs, heart, and blood makes breathlessness a thing unknown to the Styrian mountaineer, and we can readily understand the good we may obtain from this valuable drug. It is not so easy to explain how these benefits are obtained, the use of arsenic is quite empirical; like Trousseau we recognise that the patient has an increased capacity for exercise, but we cannot explain it.<sup>1</sup> "I do not know what benefit you expected from the treatment, but I know what I have received; I can go upstairs better than for many years, my breathing is easier, and my heart steadier." The old gentleman who made this remark was so sensitive to the action of arsenic that he could not bear it in larger dose than one milligramme of arsenious acid in the day (0.015 gr.); two milligrammes gave him discomfort, one was well borne. Arsenic combines well with digitalis and with strychnine, and the threefold combination is one of our most useful tonics. In sensitive patients one granule of arsenious acid containing  $\frac{1}{50}$  or  $\frac{1}{100}$  of a grain may be given once or twice a day for months or years with only increasing benefit. In combining arsenic with digitalis or with strychnine it is better to employ a fluid preparation, and the best is the *liquor arsenici hydrochloricus*, this is not only more active but in the case of combination with the *liquor strychninæ* it is the only preparation that does not make an incompatible and more or less unsightly mixture. Arsenic is a poison to which the system may be gradually habituated, so that even large doses may be taken for years not only with impunity but with positive benefit.<sup>2</sup> In those moderate

<sup>1</sup> "J'insiste sur ce phénomène éprouvé également par M. Masselot, et signalé par lui en ces termes : 'très grande aptitude à la marche.'"—*Traité de Thérapeutique*, par A. Trousseau et H. Pidoux, vol. i. p. 312.

<sup>2</sup> "On the Arsenic-eaters of Styria," by Dr. R. Craig MacLagan in the *Edinburgh Medical Journal* (September 1864), p. 200, etc.

doses in which it is given medicinally we need have no misgivings, however long it is continued. A little caution may be well employed in commencing the use of arsenic, because idiosyncrasy has a marked influence in regard to the use of this drug, and now and then we come across one who is extra-sensitive to its action. But there is not, as was at one time alleged, either risk or danger in leaving it off entirely, however long it may have been continued.

Arsenic and a bitter tonic increase the leucocytes and the number of the red corpuscles, but iron is wanted to supply hæmoglobin. For this purpose the protosalts seem to be the least disturbing to the stomach and the most easily assimilable, but in regard to this I would not be dogmatic, only it must ever be remembered that iron in combination with digitalis is extremely apt to induce sickness; it is, therefore, always best to give iron along with food, and digitalis at some other time.

In all senile hearts, and in others where the blood-pressure is high, vascular stimulants require to be combined with our cardiac tonics to avert mischief to the heart from the embarrassment due to an increase in the blood-pressure. Vascular stimulants are agents which dilate the arterioles, or at all events prevent their undue contraction, and thus keep the blood-pressure throughout the whole cardiac cycle at a uniform level, sufficient for the needs of metabolism and yet moderate enough to cause no embarrassment to the heart. All the nitrites are vascular stimulants, nitrite of sodium and spirit of nitrous ether are often used as such, but the action of the former seems not always to be safe, while that of the latter is slow and not very powerful. Nitrite of amyl is rapid in its action and is also analgesic, but it is not very lasting, the smell is disagreeable, and the flushing of the face, the fulness in the head, and the rapid action of the heart are much disliked by many patients. It is a little strange that though vascular stimulation is an action common to all nitrites, yet neither of the

two drugs that are most useful in this respect and most convenient to employ belong to this category. *Nitro-glycerine*, *glonoine* or *trinitrin*, is said to be a nitrate of glyceril, but its action is that of a nitrite. In ordinary doses of  $\frac{1}{100}$  or  $\frac{1}{50}$  of a grain it is much used to relieve the heart in angina by lowering the blood-pressure, but it is also often employed to keep the blood-pressure moderate during the use of cardiac stimulants. For this purpose it is not advisable to combine it with the cardiac stimulant, but rather to give the glonoine in sufficient dose by itself at regular intervals. The action of nitro-glycerine persists from one to four hours according to the dose, so that by giving a moderate dose every three or four hours the blood-pressure may be kept moderate and anginous attacks prevented. A 1 per cent solution acts very rapidly, and the dose is from one-half minim up to as much as ten or more minims. As tabellæ or lozenges, each containing  $\frac{1}{100}$  or  $\frac{1}{50}$  of a grain, it is more easily carried about and acts nearly as rapidly if well and quickly chewed. The drawbacks to nitro-glycerine are its liability to produce headache, giddiness, throbbing of the cerebral arteries, and palpitation of the heart, but it is remarkable how seldom these are complained of.

*Erythrol tetranitrate*, in tablets each containing one quarter of a grain, has been highly commended as a valuable vascular stimulant by Professor Bradbury and others. It is said to be more active and more permanent in its action than *trinitrin*, but is apt to produce both palpitation and headache. When more permanent action is required I prefer *iodide of potassium*, which acts as a nitrite and has the double advantage of being persistent and yet not too powerful. A couple of grains every twelve hours is quite an efficient dose, and acts admirably, more is not required for this purpose, and rarely less. The only drawback to the use of this drug is the gastric disturbance that it sometimes occasions, and then we have to fall back upon nitro-glycerine.

I need scarcely say that in syphilitic affections of the

heart iodide of potassium is our sheet-anchor, though indeed a combination with perchloride of mercury in the form of the soluble red iodide of mercury acts sometimes even better. Potassium iodide is also extremely useful in the treatment of aneurysm, but that matter I shall go fully into presently. *Colchicum* is often of the greatest service in gouty irregularity; the powder of the seeds combines well with powdered digitalis leaves, and in moderate doses both may be continued for a long time with great benefit. In such cases the alkalis-ing of the *primæ viæ* has an excellent effect, and for this there is nothing better than a tumblerful of Vichy water (source Celestins) one hour before each meal. A heaped teaspoonful of bicarbonate of potash or of soda in a small tumblerful of water acts equally well, and this thorough alkalis-ing of the *primæ viæ* often relieves irregularity in a most satisfactory manner, though it has seemed to me as if this medication favoured the formation of Heberden's knobs upon the digits.

An active purge, especially a cholagogue purge, is an excellent thing to lower the blood-pressure, and a cholagogue, by acting on the hepatic secretion, directly relieves the right side of the heart.<sup>1</sup>

Flatulence is often a disturbing element, being a frequent cause of irregularity or of *tremor cordis* in senile hearts, regulating the diet and the administration of the old-fashioned cobbler's pill—the *pilula assafœtidæ co.* of the B. Ph.—have an excellent effect.<sup>2</sup>

When severe pain is a prominent symptom there is no remedy equal to a hypodermic injection of morphine, preceded if need be by inhalation of chloroform, but for ordinary insomnia morphine is not a very suitable remedy, from the readiness with which the morphine habit is induced in some people. For ordinary use *chloralamid* in forty-grain doses

<sup>1</sup> Lauder Brunton, *Disorders of Digestion* (Macmillan and Co., London, 1886), p. 208.

<sup>2</sup> Vide *The Senile Heart*, p. 280.



rubbed up with spirit and flavoured with raspberry syrup answers very well. *Chloralalose* in doses of eight grains in cachet has this advantage that it acts quickly and induces refreshing sleep; it slightly lowers the blood-pressure without exciting the heart, and produces neither headache nor gastric disturbance; it is an ideal hypnotic for a cardiac patient. Unfortunately it has this disadvantage, that in nervous subjects it often induces exciting dreams and sometimes muscular tremors. The best way to give it is to give four grains in cachet at bed-time, this may suffice to give the patient a good night, but should he wake after an hour or two of sleep a repetition of the four-grain dose will secure a good night's rest. *Trional* has been very successful in my hands, I know of no drawback to its use. From fifteen to thirty grains at bed-time is sufficient to ensure a good night. These are the best and most reliable hypnotics in cardiac disease, sulphonal is too depressing and is so apt to postpone its action that it is not so suitable for such cases, though *faute de mieux* it sometimes acts very well in doses of five grains early in the evening and another five at bed-time. *Paraldehyde* is an excellent hypnotic and may be freely given in cases of heart complaint, the great drawback to it is its horrible taste and smell, so objectionable not to the patient only but to his relatives also. The *bromides* are not true hypnotics, merely sedatives, they are invaluable in severe and continuous palpitation of nervous origin, and by relieving symptoms they frequently induce sleep, but except in very large doses they do not act as hypnotics and cannot be trusted to.

The diet in cardiac disease is easily arranged, at least in its generalities. Those troubled with heart affections have always feeble digestion, partly from the gastric venous congestion present, and partly from a gastric juice defective both in quantity and quality, hence we must restrict the quantity of food, see that the gastric juice is not further impaired in quality by dilution, and lastly we must secure a

sufficient interval between each meal. In health the stomach requires from three to four hours to complete the digestion, and it needs an hour's rest before it takes in a further supply. In all with feeble circulation digestion is slower than in health, we must therefore allow fully four hours for digestion and one hour for rest, making the interval between one meal and its successor not less than five hours. During the period of digestion no solids must be ingested, as the introduction of even the smallest bit of biscuit under these circumstances arrests digestion and is a common cause of flatulence. It is also of consequence that those with feeble or diseased hearts should have their principal meal in the middle of the day, and that all their meals should be as dry as possible; when thirst is complained of hot water—a most excellent cardiac stimulant—may be taken to the extent of a pint one hour before each meal. These are the general rules upon which we must act, but each particular patient must be catered for by himself, and it is impossible to lay down one hard and fast dietary applicable to all.<sup>1</sup>

Considering the difficulty most cardiac patients have in making the slightest exertion, it seems almost superfluous to insist upon the necessity of rest, yet this is a matter of paramount importance only too apt to be neglected by the patient himself, whose days are often brought to an untimely end by ill-judged and injurious exertion. Whenever we have to do with an uncompensated or imperfectly compensated valvular lesion, whenever, in short, the myocardium is from any cause debilitated, rest is imperatively indicated. The heart does not differ from other muscles which require rest when suffering from exhaustion,<sup>2</sup> you must therefore never forget to insist upon rest as an important part of the treatment, rest which must be more perfect and complete

<sup>1</sup> A more detailed statement as to diet, chiefly as applicable to one form of heart affection, will be found at p. 236 and following pages of the *Senile Heart*.

<sup>2</sup> Lauder Brunton, *Lectures on the Action of Medicines* (London, 1897), p. 304.

the more serious the cardiac disturbance is. Rest in the recumbent posture means a reduction in the pulse-rate by an average of 12 (6-16) beats per minute, and in weakly ill-fed patients the reduction is sometimes greater. This lowering of the pulse-rate amounts cumulatively to an increase of rest to the heart of over two hours in the twenty-four, and we can readily understand that this increased rest, coupled with warmth and better feeding, is of itself sufficient to give such a fillip to the general metabolism as enables the heart to recover its tone and to discharge its function more perfectly. The blood-pressure rises, the balance of the circulation as between arteries and veins is restored, dropsy disappears, and the patient is re-established in comparative health by the aid of rest alone coupled with a favourable change in his environment. But though for his heart's sake we keep our patient recumbent and at rest, we do not let his muscles waste, we prevent this by massage, which is of itself both a help to the circulation and an aid to general metabolism.<sup>1</sup> But cardiac patients are not to be confined to bed or to a Bath chair for the natural term of their life. We have constant opportunities of seeing that well compensated cardiac disease is not incompatible with even strenuous exertions. And we have also frequent opportunities of observing that after rest and appropriate treatment have improved the heart's nutrition and energy, and compensation has been restored, the patient re-acquires considerable aptitude for exertion. And as, if metabolism be perfect, every organ improves by the exercise of its function, so also a weak, feeble heart, or even a heart affected with a compensated valvular lesion may be much benefited by moderate and regulated bodily exertion. This is a doctrine first

<sup>1</sup> "Over all Polynesia, and a part of Micronesia, the rule holds good; the great ones of the isle, and even of the village, are greater of bone and muscle, and often heavier of flesh, than any commoner. The usual explanation—that the high-born child is more industriously shampooed—is probably the true one. In New Caledonia, at least, where the difference does not exist or has never been remarked, the practice of shampooing seems to be itself unknown."—R. L. Stevenson, *In the South Seas: Travels and Excursions*, vol. iii. p. 81.

enunciated by Stokes, who recommended graduated exercises in the treatment of those weak hearts which he thought to be fatty.<sup>1</sup> If we are to believe a personal reminiscence of von Ziemssen, Stokes even went so far as to compel a sufferer from aortic incompetence to run behind his own carriage, a proceeding which none of us are likely to imitate.<sup>2</sup> Thus you see that though rest is an important therapeutic agent, yet there comes a time when exercise is not only not to be debarred, but may be made a most useful adjunct to other treatment. Each case, however, must be differentiated and treated on its own merits, and to propound graduated exercises, with or without any form of baths, as a panacea for all forms of heart affection is to court certain disaster. The neat little diagrams we so often see propounded as proofs of the diminution in size of a dilated heart after a bath are most delusive. The heart is so mobile an organ, and varies so incessantly in size (*note*, p. 17), that any attempt to define its degree of dilatation by percussion alone, auscultatory or otherwise, cannot but be entirely untrustworthy.

When considerable dropsy is present it may be removed by the kidneys, by the bowels, or by tapping. In cardiac dropsy digitalis is a most efficient remedy properly employed; various combinations with squill, with mercury, or other drugs have been used, but digitalis alone is quite sufficient. Purgatives are trusted to by some, and the electuary of the bitartrate of potass is an excellent adjunct to digitalis, or it may be trusted to alone; others prefer more drastic purges, such as croton-oil, or elaterium, or a combination of both in moderate doses given every four hours till free catharsis is induced. When the limbs from anasarca are firm and tense, either purgatives or drainage must first be resorted to, to remove the peripheral obstruction that hinders the action of

<sup>1</sup> *Diseases of the Heart and Aorta* (Dublin, 1854), p. 357.

<sup>2</sup> *Verhandlungen des Congresses für innere Medicin* (Wiesbaden, 1888), S. 55.



digitalis,<sup>1</sup> as Withering pointed out more than a century ago (1775). Caff  ine has been highly recommended, of fair value as a diuretic; it is occasionally useful but cannot be relied upon. On the other hand, in diuretin (sodio-theophyllinate) we have a powerful diuretic which in a dose of 15 grains every three or four hours is hardly known to fail; it is best given dissolved in hot water and allowed to cool. The single dose is 15 grs., the maximum quantity in one day should never exceed 120 grs., it need seldom amount to so much.

Occasionally, however, it is desirable to relieve the patient rapidly and certainly, and this is most effectually done by drainage. The fluid in an uncomplicated heart case is pure serum and does not irritate the skin like that effused in kidney cases, we need not therefore hesitate to make sufficiently free incisions to let the fluid escape. In this way drainage proceeds rapidly, and the wounds if kept aseptic heal quickly. Southey's tubes keep the bedding dry, but that can be otherwise provided for. From the small calibre of these tubes drainage is slow, and if they are used it is well to have a larger size of tubing attached to the cannula than is ordinarily supplied.

<sup>1</sup> *Vide antea*, p. 366.

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## LECTURE XV

<sup>1</sup> ON THE SIMULATION OF ANEURYSM BY MALPOSITION OF THE  
 AORTA, DUE TO RICKETS

SOME years ago a young man presented himself to me, stating that he had been under treatment for about six months for supposed aneurysm of the aorta, and was desirous of having my opinion as to his present state and future prospects. Upon stripping this patient, inspection at once revealed that he had an abnormal pulsation between the second and third ribs on the right side, extending for about three-quarters of an inch to the right of the sternum. It was also evident that he had a slight scoliosis (lateral curvature) of his spinal column. Upon palpation the chest was felt to expand a little more freely on the right than on the left side; the abnormal pulsation was felt to be distinctly fluid in character, synchronous with the apex beat, and not more forcible. Upon mensuration at the level of the fourth rib, the right side of the chest was found to measure a full inch more than the left; at full expansion the right side measured fourteen inches and a half, and the left thirteen inches and a half. Percussion of the chest revealed nothing unusual except slight dulness over the abnormal pulsation to the right of the sternum, a dulness that was strictly limited to the pulsation and did not extend beyond it. Auscultation also revealed nothing unusual; the cardiac and pulmonary sounds were perfectly normal. Over the abnormal pulsation nothing was to be heard but the sounds normally heard over the aortic area. Moreover, there was an entire absence of

any signs of abnormal pressure, or even of any symptom of such a thing. The young man was in perfect health, of fair physical development, and he had felt quite equal to any exertion up to the time when he was put under treatment. From a due consideration of all the facts elicited on examination, and thus cursorily narrated, I had no difficulty in assuring the young man—he was no patient—that he had no aneurysm, that he was in perfect health, fit for any occupation, and only the subject of a trifling malformation. I need not say how thankful and pleased he was. A year subsequently he was passed into the army. When I last saw him his chest had considerably developed, and though the relative size of the two sides of the chest remain identical (right 18, left 17), yet the artery was better covered, and the abnormal pulsation was no longer to be felt.

This case is not unique; there are many such. Very possibly there may have been some similar cases whose lives have been embittered, and whose friends have been made unnecessarily anxious by an unguarded prognosis; and to enable you to avoid this error is the object of the present lecture. Hitherto these arterial malpositions have been—from a physician's point of view—entirely overlooked.<sup>1</sup> From an anatomico-pathological point of view, such cases have, however, been long well known, and Morgagni,<sup>2</sup> Watzel,<sup>3</sup> Vrolik,<sup>4</sup> Otto,<sup>5</sup> Tiedemann,<sup>6</sup> and others have recorded many interesting cases. More recently the whole subject has been

<sup>1</sup> The earliest reference to the diagnostic importance of aortic malposition, so far as I know, is contained in a short paper by myself in the *Edinburgh Medical Journal*, February 1871, entitled "Cases illustrative of some Difficulties in the Diagnosis of Aneurysm close to the Heart," at p. 707.

<sup>2</sup> *De Sedibus et Causis Morborum*, Ebroduni in Helvetia (1779), 4.

<sup>3</sup> *De efficacia Gibbositatis in Mutandis Vasorum Directionibus*, Trajecti ad Viadrum (1778).

<sup>4</sup> *Dissertatio de Mutato Vasorum Sanguiferorum Decursu in Scoliosi et Cyphosi* (Amstelodami, 1823).

<sup>5</sup> *Lehrbuch der pathologischen Anatomie* (1830).

<sup>6</sup> *Supplementa ad Tabulas Arteriorum Corporis Humani* (Heidelbergæ, 1846). Hartmann, Cheselden, Ludwig, and Wenzel have also described these alterations in the course of the vessels, from a pathological point of view.

gone over from a similar point of view by Dr. Barkow, Professor of Anatomy at Breslau, who has figured numerous examples of this interesting peculiarity.<sup>1</sup> One of the most remarkable of these is contained in the fourth plate of his earlier work. The accompanying woodcut is reduced from this figure; in it the ascending aorta is seen to reach the surface of the chest between the third and fourth, and the second and third ribs on the right side; while the descending

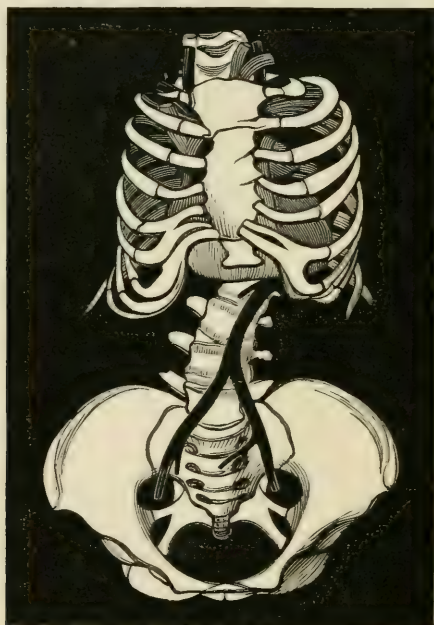


FIG. 31.

aorta gets to the surface between the second and third ribs on the left side, the convexity of the scoliosis being to the left.

In the case of the young man just narrated, the scoliosis was so slight, that in all probability the abnormal exposure

<sup>1</sup> *Die Verkrümmungen der Gefäße dargestellt*, von H. C. L. Barkow, M.D., etc., Fol. (Breslau, 1869), and *Erläuterungen zur Lehre von den Erweiterungen und Verkrümmungen der Gefäße*, von H. C. L. Barkow, M.D., etc., fol. (Breslau, 1871).



of the vessel was due to some slight malposition of the aorta, rather than to any deflection of that vessel caused by the trifling curvature of the spine that existed. This, however, rather enhances the importance of the case from a diagnostic point of view. For while it is of consequence to remember that in rickety chests the aorta may be so deflected, without any marked dilatation, as to make its pulsation visible either to the right or left of the sternum, and so to simulate an aneurysm, it is of even greater consequence to have proof that in certain comparatively rare cases a similar abnormal pulsation may be due to a trifling divergence from the normal course of the vessel itself, apart from any marked change in the bony skeleton. But we must never forget that aortic aneurysm may coexist with malformation of the thorax, with or without scoliosis; and whatever may be the condition of the skeleton, any abnormal pulsation must be carefully considered from every point of view, before we are able to give any definite opinion as to what it really is. For the art of diagnosis is based upon an estimate of probabilities, and its approximate accuracy depends, first, upon the accuracy with which we make our preliminary observations, and, second, upon the intelligence and capacity employed in our reasonings regarding these observations. For example, any cursory observer who had seen, but not duly considered, the case just narrated, and who shortly afterwards was brought face to face with the woman Murray, now in Bed 15, Ward XIII., would probably feel little hesitation—might even plume himself on his sagacity—in placing both in the same category; and yet he would thereby commit a most egregious blunder. It is true that Murray has an abnormal pulsation between the second and third, and the third and fourth ribs on the right side close to the sternum. Her left chest also measures a full inch less in circumference than the right one. She has no scoliosis, but in the young man this was so slight as to be unimportant, and in either case the diagnosis turns entirely upon quite other points, to which I shall presently

direct your attention. I shall first relate to you the histories of two extremely interesting cases, in which aortic aneurysm was closely simulated by vascular deformity depending upon rickety malformation of the thorax. Having already pointed out those negative signs which determine the diagnosis in the case first referred to, I shall conclude this chapter with a cursory indication of those positive signs upon which we rely for the diagnosis of aortic aneurysm. A due consideration of these signs enabled us to arrive at a very different conclusion in regard to the case of the woman Murray.<sup>1</sup>

CASE XXXVIII. G. S., a shoemaker, aged thirty-five, admitted to Ward V. on 5th December 1870, complaining of breathlessness, cough, spit, and occasional hæmoptysis. When about sixteen years of age, while acting as a cattle herd, he was occasionally troubled with pains in his shoulders. But he has neither had rheumatism nor any other disease since that time, with the exception of a slight attack of ague (in Iowa, U.S.A.) about two years ago. When eighteen years of age he suffered from loss of appetite, and occasional attacks of squeamishness and faintness. At this time he had been about two years engaged at shoemaking; he was well fed, and was able to remain at work, but just at this time his thorax became gradually deformed.

On admission his spinal column was found to have a double lateral curvature—the thoracic curve being to the right, and the compensatory dorso-lumbar curve to the left—while the lower dorsal and lumbar vertebræ were so twisted that their transverse processes lay somewhat diagonally. The ribs were broad and flat, the upper ones apparently wider apart than usual, while those below the sixth were crowded together. The anterior part of the thorax was so altered that the right side was round and prominent, while the left was flattened and compressed. The lower part of

<sup>1</sup> This patient subsequently died, and her aneurysm was exhibited at the Edinburgh Medico-Chirurgical Society, on 3rd May 1876, vide *Edinburgh Medical Journal*, June 1876, p. 1141.

the sternum was concave, as is usually the case in cobblers. Both feet were twisted outwards, but they were said to have been so from infancy.

About a year previous to admission, he began to have, at times, a severe pain across the front of his chest below the *mammæ*. This pain came on while stooping over his work, but also when otherwise engaged, occasionally even when in bed, and this it did sometimes for two weeks at a stretch, compelling him continually to shift his position, and even to get out of bed. He did not think that any change of posture gave relief. This pain came on suddenly, was not accompanied by any breathlessness, and it usually went away quite as suddenly as it came; he attributed it to the pressure of the boot or last on his breast, necessary in his trade.

About six months previous to admission, this patient first began to have shortness of breath, at first only felt on making any sudden movement, such as raising himself quickly from a stooping posture; but it speedily came to follow any exertion, however slowly made, especially such as ascending a hill, etc. During the summer of 1870 he had considerable cough, and in August of that year, when bathing, he brought up some blood mixed with the sputa; this trifling hæmoptysis lasted only one day. But his cough got worse, and six weeks later his hæmoptysis recurred and lasted for three or four days. About one month before admission he had a third attack of hæmoptysis, which lasted for three weeks.

On inspection of the thorax, the points already indicated were seen, and considerable heaving of the præcordial region was also noted, the spaces between the fourth and fifth, and fifth and sixth ribs being retracted at each diastole. Between the fifth and sixth ribs lay the lowest point of cardiac pulsation to be seen or felt; beneath this the ribs closed up, they almost overlapped each other, and extended down into the pelvis. The cardiac pulsation referred to was, however, no apex beat, but a broad impulse extending over a space of two inches and a half. The large arteries at the root of the neck pulsated

visibly with great force, and a thrill was felt with each pulsation. On laying the hand across the upper part of the chest considerable thrill was felt, chiefly towards the right edge of the sternum and over its upper part. On placing the finger in the tracheal fossa, the aorta was felt pulsating within half an inch of the upper edge of the manubrium. Between the second and third ribs, on the right side, a pulsating tumour was felt extending for about an inch to the right of the sternum.

On percussion on the left side, one inch from the sternum, the note was clear down to the upper edge of the fourth rib; beneath this there was dulness to the upper edge of the sixth rib, and beneath this only the tympanitic note of the stomach was to be heard. In the nipple line (level of the fourth rib) dulness commenced about one inch to the right of the sternum, and extended to the left for a distance of four inches and three-quarters. Along the right side of the sternum, dulness extended from the upper edge of the second rib down to the liver dulness for a distance of one inch to the right of the sternum.

On auscultating over the lowest perceptible part of the cardiac impulse, the first sound was heard somewhat muffled, and the second was replaced by a murmur. Between the second and third ribs at the right edge of the sternum, a loud rough murmur wholly replaced the first sound, and a soft blowing murmur completely occupied the time of the second sound. Both of these murmurs were loud and harsh over the pulsating tumour already referred to, and they were softer in character, though equally distinct, on auscultating over the sternum. These murmurs were propagated into the arteries in the neck, and across the sternum to the left. The pulmonary second was distinctly audible just over the left edge of the sternum close to the second interspace; within that space this second sound was only faintly to be heard quite close to the sternum.

The patient's pulse was 86, full and jerking; it was also



delayed, the radial beat coming just between two cardiac impulses and as nearly as possible equidistant from both. On admission his cough was nearly gone, but he still had a slight amount of purely catarrhal expectoration. The other phenomena were unimportant—either natural, or without bearing on the case.

In this case the whole of the urgent symptoms and signs were those of incompetence of the aortic valves, a somewhat rare occurrence in a case presenting so many indications of a sacculated aneurysm just above the valves. For when the disease has originally been a sacculated aneurysm of the aorta the heart is rarely much implicated, even after incompetence of the aortic valve has been superadded. Further, the signs present—especially the slight amount of dulness to the left of the sternum, the great amount of dulness to the right of that bone, and particularly the fact that the pulmonary second was scarcely audible at all to the left of the sternum, and was only to be heard distinctly after the stethoscope had been placed upon that bone—all pointed to the great probability that the heart had been slightly dislocated to the right. This idea, if correct, would sufficiently explain the appearance of a pulsating tumour between the second and third ribs on the right side, and extending not more than one inch from the right margin of the sternum, as this is the precise position that would be occupied by the aorta in such a case. The probability that the pulsating tumour was of this character was further increased by the discovery, from the augmented dulness across the upper part of the sternum and from the pulsation in the tracheal fossa, that the transverse portion of the aorta was dilated; because all experience teaches us that in a case of aortic incompetence with a dilated transverse portion, dilatation of the ascending part of the aorta is almost certain to coexist. But any dislocation of the heart to the right would suffice to push even a normal aorta from under cover of the sternum, and still more, therefore, one which is dilated. Moreover, the slight

increase in loudness of the abnormal murmurs, in this case, as heard over the pulsating tumour, was no greater than was to be expected from the closer proximity to the ear of this pulsation, as compared with an artery lying beneath the sternum, and had none of that marked accentuation which these murmurs gain when heard over the sac of an aneurysm. Pressure symptoms were also entirely wanting. The lung on both sides was displaced, and to this was due the diastolic dimpling of the fourth and fifth interspaces on the left side; and the greater loudness and roughness of the murmurs over the tumour than over the sternum was undoubtedly due to the contiguity of the aorta to the chest wall.

The slight displacement of the heart was evidently due to the deformed condition of the chest produced by rickets. The base of the heart was more displaced than its apex, as if the enlarged heart, resting on the diaphragm in this deformed and stunted body, leaned somewhat forwards and to the right into the bulging right half of the thorax; and this I have no doubt it did.

This patient died on 1st January 1871 from acute oedema of the lungs. A proper examination of the body was refused by the friends, but I was enabled to ascertain satisfactorily that the aorta was dilated and displaced to the right, as already indicated, and that no sacculated aneurysm was connected with it.

There can be no doubt that the cardiac disease, in this case, was primarily due to the obstruction opposed to the circulation by the sinuosity of the arterial trunks caused by the distortion of the skeleton. And we scarcely need the experience and assurance of Barkow to convince us that obstruction of this character has, and must have, an unmistakable influence in promoting dilatation and hypertrophy of the heart.<sup>1</sup> The larger blood-wave and the increased energy of the heart have a special influence in producing dilatation of the aorta, especially of its ascending portion,<sup>2</sup>

<sup>1</sup> *Die Verkrümmungen der Gefäße*, S. 15.

<sup>2</sup> *Op. cit.* S. 35.

an influence which, as we can readily understand, may be very considerably modified by the structural condition of the aorta itself.<sup>1</sup> When once dilatation of the ascending aorta is produced, the secondary development of incompetence of the aortic valve is merely a question of time.

The case now to be narrated was repeatedly under observation, and was always an object of great interest.

CASE XXXIX. M. P., a milliner, unmarried, aged forty-six, admitted to Ward XIII. on 26th January 1871, complaining of cough, feeling of oppression over the trachea, and of general debility. Patient had been a milliner for thirty-three years, working during the busy season from 6 A.M. of one morning to 1 A.M. of the next. As the result of this overwork she was never strong, and never free from headache; still she could take her food well, and was able to continue at work. About sixteen years before admission, she first noticed that her right shoulder was somewhat distorted and her body also somewhat twisted. Since that time this distortion has slowly increased, but the change has been gradual, and she has suffered from no illness more severe than a casual and temporary catarrh, till seven years ago, when a cough then acquired became chronic, and has never entirely left her. When this cough first affected her, she was neither ill nor feverish and was unconscious of having caught cold. Two years subsequently this persistent cough was aggravated by an attack of bronchitis, which lasted five weeks, and broke down her health very much. Since that time her distortion has steadily increased; and about four years ago her bodily distortion and weakness became so great that she had to give up her occupation. Since then she has been constantly subject to a dull, aching pain, referred to the right shoulder-blade, the intensity of which has gradually increased. She has also been subject to pain in the region of the stomach, more or less constant, but worse when the stomach is empty, and somewhat relieved by taking food.

<sup>1</sup> *Op. cit.* S. 15.

For some years she has been aware of a pulsation in the front of her neck, but her attention was not particularly directed to it till the morning of 23rd January 1871, when, after a severe fit of coughing, she accidentally observed that where this simple pulsation formerly existed, there was now a throbbing swelling as large as a hen's egg. A feeling of tightness and choking referred to the chest and lower part of the throat, which subsisted after the cessation of the paroxysm of coughing, caused her to look at her throat and so to discover this pulsating tumour. On admission the patient was seen to be much emaciated and etiolated. There was considerable scoliosis of the spinal column in the dorsal region, with right-side convexity, some protrusion backwards, and a compensatory lumbar curve to the left. On the right side the thorax was thrown outwards and backwards, and flattened laterally; on the left side the ribs were indented and compressed together.

On palpation the only pulsation to be felt on the left side was between the third and fourth ribs about half an inch to the left of the sternum. To the right of the sternum pulsation was found in the first, second, and third interspaces. This pulsation was most forcible and distinct in the second interspace, where it extended a couple of inches to the right of the sternum, and in this situation considerable thrill was to be felt. In the lower part of the neck, just over the suprasternal notch, a pulsating tumour was felt crossing the trachea and dipping beneath the right sterno-cleido-mastoid muscle; this was evidently a dilated vessel of abnormal distribution; it was apparently continuous with the right subclavian artery, which was large and dilated, as was also the brachial. The left subclavian was also dilated. The aorta was not felt on passing the finger deep into the tracheal fossa. All the arteries at the root of the neck pulsated strongly.

On percussion cardiac dulness, at one inch from the sternum on the left side, was found to commence at the upper border of the second rib and extended down to the



liver dulness. From the upper border of the second rib on the left dulness passed obliquely across the sternum to the lower border of the first rib on the right, and between the first and second ribs (in the first interspace) to the right of the sternum the dulness passed to the right for a distance of two inches. It also passed downwards to the liver dulness in the same parasternal line. At the level of the fourth rib the transverse dulness was four inches and a half. On auscultating over the cardiac pulsation felt between the third and fourth ribs, half an inch to the left of the sternum, both sounds of the heart were to be heard, but neither of them pure. Over the fifth rib, one inch from the left edge of the sternum, these sounds were to be heard with greater distinctness and purity, but no pulsation was to be felt. In the left second interspace, distinct but impure first and second sounds were audible. In the second interspace to the right of the sternum the first sound was impure and the second was obscured and almost entirely replaced by a diastolic murmur. Over the pulsating tumour already referred to as chiefly lying between the first and second ribs to the right of the sternum, where the pulsation was most fluid and most forcible, there was a loud systolic murmur followed by a less distinct diastolic murmur. These two murmurs had their position of maximum intensity in this situation, and from it both radiated outwards in all directions. The systolic murmur was propagated upwards with most distinctness, and the diastolic murmur downwards, but less distinctly *pari passu*, being more faint *ab origine*. The percussion of the lungs was equal on both sides, and the respiration was also equal, though neither could be held to represent an average normal, as the percussion was slightly higher in pitch, and the respirations slightly rougher in character than usual. No other symptom of any importance was observed, nor was any other sign present except a considerable thrill, which was perceptible over the pulsating tumour already described.

The diagnosis in this case was not very difficult; the absence of the slightest indication of any abnormal pressure was quite conclusive as to there being neither a sacculated aneurysm nor a cirroid bulging of arterial origin in the region of the abnormal pulsation in the first interspace to the right of the sternum. The absence of pressure symptoms also clearly indicated that this pulsation was simply arterial in character, and this was further confirmed by the strict limitation of the dulness to the pulsation. But a pulsating artery in the region referred to could only be the aorta, and if this pulsation was only a dilated aorta it must be of unprecedented dimensions to present so large a superficial area of dulness. The position of the cardiac pulsation, however, as well as the situation and form of the area of cardiac dulness, assured us that the heart was tilted upwards as well as thrown more to the right of the sternum than usual. Hence we had the ascending portion of the aorta passing more directly outwards to the right, and as the transverse part of the arch did not rise higher nor take a wider sweep than usual, it must necessarily have made an unusually acute angle at its junction with the ascending part of the aorta. But compression of the arterial lumen at this acute angle was sufficient to account for the systolic thrill and the loud systolic murmur, both being due to the formation of fluid veins at this constricted part. As for the diastolic murmur, in the absence of any saccular aneurysm, which we did not believe to exist, this could only be due to regurgitation through the aortic valve. This regurgitation would be the necessary result of the separation of the valve segments by hydraulic pressure of the blood accumulated in the dilated ascending aorta. This inevitable physical result might, however, be complicated and hastened by the certainly less inevitable physiological result of pressure—local endocarditis with thickening and shrivelling of the valve segments.

This patient died suddenly, in Ward XIII., on 7th November 1872. At the autopsy the lungs were found

congested and œdematous. The heart was tilted up, and the great vessels displaced. The ascending aorta passed outwards to the right more than usual, and the transverse part passed off from it at a somewhat acute angle. The innominate artery was two inches long and twice its usual diameter. It came off from the aorta further to the left than usual, coming to the surface at the left sterno-clavicular articulation, and passing across the trachea in the lower part of the neck to the edge of the right sterno-cleido-mastoid muscle, beneath which it dipped and divided. The aorta was slightly atheromatous and dilated at its commencement, but perfectly free in every part from any saccular enlargement. The aortic valve was slightly incompetent; its segments were thickened. The heart itself was hypertrophied and dilated, particularly on its right side, but to no great extent. The mitral valve was competent, but had a few vegetations on its upper surface. The tricuspid valve was healthy; its opening admitted five fingers. The pulmonary valve was healthy and competent. The abdomen was filled with fluid; the liver was slightly enlarged, and the kidneys in a state of chronic congestion. No other organ was examined.

This case was thus another example of rickety distortion of the skeleton giving rise to abnormal conditions of the blood vessels closely simulating aneurysm. Of this simulation the case now before us may be regarded as a somewhat extreme example; and between it and the case narrated at the commencement of this lecture there are infinite gradations, examples of which are of occasional, though not of very common occurrence. It is well to be aware of the occurrence of such cases, so that we may be prepared for them, and thus avoid falling into such a mistake as happened in the case with which I commenced; the anxiety and worry connected with it were very serious to the poor man himself and to his friends, and the unfortunate mistake might very well have proved tragic to all concerned.

It is well, therefore, to remember that even a fluid

pulsation in any of the intercostal spaces is not necessarily an aneurysm. The absence of any history of empyema, and even the distinct connection of the pulsation with the aorta by continuity of dulness, is no proof of its aneurysmal character, either in the sense of a saccular aneurysm or of a mere dilatation. Even in the normal condition of the skeleton the aorta may be exceptionally so deflected as to permit its pulsations to become perceptible in one or other of the intercostal spaces; while abnormal intercostal pulsation of simple arterial origin is a matter of no infrequent occurrence whenever the thoracic skeleton is deformed by rickets. Distinctly saccular aortic aneurysms may also, of course, occur even in chests deformed by rickets.

When there is no twisting or bending of the artery, and no aortic incompetence, the case first narrated seems to show that—as we would expect—there is no murmur audible over the abnormal pulsation, and the only sounds audible are those normally heard at the base of the heart, the second in particular being in no degree accentuated. Whenever a murmur of regurgitation is developed at the base of the heart, that is always more or less audible over the ascending and transverse portions of the aorta. Apart, also, from any obstruction or constriction at the mouth of the aorta, we are sure to have a systolic murmur developed over any part of the artery where any sharp twist or bend occurs. Moreover, as any abnormally situated intercostal pulsation must be nearer the surface than any normally situated part of the artery, all sounds and murmurs are more distinctly heard over that pulsation than elsewhere, yet without accentuation. Whenever we perceive marked accentuation of the murmurs or sounds present, and especially if the normal second sound be markedly accentuated, we are bound to suspect the presence of a saccular aneurysm, notwithstanding the presence of rickety malformation. To make our diagnosis certain, however, we must be able first of all to connect the pulsation directly with the aorta, and, secondly, we must be able to



show that the dulness subtended by this pulsation extends beyond its limits, and thus occupies a space greater than if the pulsation were due to a simple cylindrical vessel like the aorta in contact with the chest wall. Extension of dulness beyond the limits of the pulsation, associated with signs and symptoms of pressure upon one or more of the neighbouring organs, are among the most certain indications of the presence of an aneurysm, whether saccular or cirroid. Associated with these signs and symptoms we also have other phenomena that indicate the dependence of the pressure upon an elastic and distensile body of varying dimensions, and by due attention to all the facts elicited we are often able to prognosticate the existence of an aneurysm even when no pulsating tumour has been detected, and will also be able to determine with perfect accuracy the true character of any pulsating tumour that may be perceptible. But I must reserve full consideration of this matter till next lecture.

## LECTURE XVI

### ON THE DIAGNOSIS OF AORTIC ANEURYSM, ITS COURSE AND TREATMENT

THE diagnosis of thoracic aneurysm is sufficiently obscure at times, while at others it seems so patent that it is difficult to conceive the possibility of a mistake.

I need hardly say that an aneurysm is a local dilatation of an artery; of all its coats in a fusiform or globular shape, or a mere bulging of these coats on one side of the vessel; both of these forms are often combined in the thoracic aorta, in which uniform dilatation is frequently associated with local bulgings. An aneurysm caused by uniform dilatation of all the coats of an artery is called a *true aneurysm*. Any mere bulging on the side of an artery is often entirely unaccompanied by any general dilatation of the vessel, but accompanied and apparently caused by rupture of one or other of the arterial coats; this is what is called a *false aneurysm*. It is also often termed a *saccular aneurysm*; but it is well to remember that this term is also frequently applied to those local bulgings occasionally associated with a so-called true aneurysm (simple dilatation), especially when such a bulging is solitary. During life it is scarcely possible to state, even with a minimum of probability, whether a saccular aneurysm is true or false. But it is quite possible to say with great probability whether it is associated with dilatation of the artery, and therefore probably a mere bulging, or whether it stands alone, and is therefore more likely to be not only a saccular but also a false aneurysm.

False aneurysms have been subdivided pathologically into several varieties, according to the number and the nature of the coats ruptured; clinically this subdivision is a matter of no importance, and impossible to recognise. So-called *dissecting aneurysms* are formed by rupture of the internal and middle coats with effusion of blood either between the layers of the *tunica media*, or between the *media* and the *adventitia*. The diagnosis of such an event is not easily made; fortunately this is of little consequence so far as treatment is concerned, though it might be of importance as regards prognosis. *Varicose aneurysms* chiefly affect the arch of the aorta, and are caused by abnormal communications between the aorta and the superior vena cava, the pulmonary artery, or the right auricle. These give rise to phenomena of great pathological interest, are usually rapidly fatal, and are not amenable to treatment. *Cirroid aneurysm* of the aorta is common enough if we accept Rokitsanski's definition; he says that any cylindrical or fusiform dilatation of the artery accompanied by an apparent increase in the length of the vessel, and by saccular bulgings first on one side and then on the other, so that the vessel appears to wind from side to side, and lies in apparent coils, is entitled to this appellation.<sup>1</sup> A great number of so-called true aneurysms of the thoracic aorta present this appearance, and are therefore entitled to be called cirroid, though they have nothing in common with the external so-called aneurysms usually recognised by this term.

Again, when we speak of aneurysms of the aorta we ordinarily refer to those occurring above the semi-lunar valves, but it would be wrong to regard these as the only aneurysms of the aorta, for, besides aneurysms of the valves themselves—which ought, strictly speaking, to be looked upon as belonging to the aorta, but which only reveal themselves as valvular lesions—we have also aneurysms immediately within the valves and above the cardiac ventricle

<sup>1</sup> *Handbuch der pathologischen Anatomie* (1844), Bd. ii. S. 551 and 557.

(intra-valvular aneurysms), and aneurysms between the valves (inter-valvular aneurysms), the latter form being the most rare; but both forms are exceedingly uncommon.<sup>1</sup> The symptoms during life, so far as recorded, were only those of valvular lesion; clinically, therefore, they possess no peculiar interest, though they are of considerable pathological importance.

True aneurysms—simple dilatations of whatever form—whether accompanied or not by bulgings, and false or saccular aneurysms, arising above the valves, are those of the greatest clinical importance, and they are more frequent in the thoracic aorta than in any other part of its course. This we learn conclusively from Dr. Sibson, who at great personal trouble has collated the records of 584 cases of aneurysm, and has also examined 296 specimens in museums, to which no histories are attached—880 cases in all.<sup>2</sup> From Sibson's researches the following table has been compiled:—

Situation of Aneurysm.	Total Number.	Percentages.		
		Of these there were sacculated.	Mere Dilatation of Vessel.	Character of Aneurysm not defined.
Sinuses of Valsalva . . . .	87	95·95	3·56	
Ascending Aorta . . . .	141	54·75	38	7·25
Do., Dissecting Aneurysm . .	52			
Transverse Aorta . . . .	120	44·25	20	12·5
Ascending and Transverse Aorta conjointly . . . .	112	40·7	85·7	
Descending portion of Arch . .	72	71·9	14·55	17·7
Transverse and descending portion of Arch . . . .	20			
Whole Arch . . . .	28			
Descending Thoracic Aorta below Arch . . . .	71	42·4	42·55	20·4
Abdominal Aorta at Coeliac Axis .	131	70·35	11·85	18·4
Do., below Mesenteric Artery .	26			
Branches of Abdominal Aorta .	20			

Thus of 880 cases of aneurysm no fewer than 703 belong to the thoracic aorta, and if we take any one portion of its

<sup>1</sup> Two cases of intra-valvular and one of inter-valvular aneurysm have been published by Rokitsanski in the *Medizinisches Jahrbuch* (Wien, 1867), S. 174.

<sup>2</sup> *Medical Anatomy* (London, 1869), columns 57 and 58.



course we find that by far the larger number belong to the ascending aorta, which comprises no fewer than 193, 52 of these being dissecting aneurysms. The transverse part of the arch is much less frequently involved, its aneurysms numbering only 120 out of the 880. Aneurysm of the descending aorta is still more rare, only 72 out of the 880 belonging to that part of the artery. Further, if we take the number of aneurysms affecting the ascending and transverse portions conjointly, and contrast them with the numbers arising from the transverse and descending portions of the arch conjointly, we find the relative numbers to be 112 and 20. These facts show the important relation the ascending part of the aorta bears to aneurysm, evidently due to the strain from the blood forcibly propelled from the ventricle being most severely felt in this situation.

The middle coat is that upon which the strength, firmness, and elasticity of the arterial tube depends, and loss of elasticity in it must precede, and is the primary cause of arterial dilatation or aneurysm, the secondary and effective cause being of course the intra-arterial blood-pressure. Local bulgings and saccular aneurysms are primarily due to local atrophies or ulcerations of the middle coat. These affections are degenerative in character, and are usually described as following a primary affection of the *intima* (*endarteritis chronica*); sometimes, however, the *media* itself is primarily affected, and more rarely aneurysms have been found to follow a circumscribed inflammation implicating the external and middle coats (*periarteritis nodosa*), or the impaction of a sharp pointed embolus. Falls, blows, and violent exertions are often the direct cause of an aneurysm, by rupturing the coats of a vessel already diseased.

There is no disease the diagnosis of which is more beset with difficulties than thoracic aneurysm. But there is also probably no disease in which a due consideration of symptoms and physical signs, as well as their modification by position, exertion, etc., and of the mode in which they have been

primarily developed and have subsequently progressed, is more capable of conducting us to a satisfactory, if not always an absolutely certain diagnosis. There is only one phenomenon positively characteristic of thoracic aneurysm, and that is—*The existence in some part of the thorax of a pulsating tumour other than the heart, which beats isochronously<sup>1</sup> with the heart and at least as forcibly, and which at each pulsation expands in every direction.* These signs clearly recognised are sufficiently distinctive, and yet they are occasionally so efficiently simulated as to necessitate great care in their determination.

*A solid tumour* lying on the aorta may by compression originate a murmur and may appear to pulsate, but it only rises and falls with the beating of the artery, and does not expand in every direction.

*A vascular tumour*, be it mediastinal or pulmonic, may possess not only a murmur but also a certain degree of expansive pulsation; that pulsation is not, however, *isochronous* with the heart's action, but is always somewhat delayed.

*An empyema* may present both bulging and expansile pulsation isochronous with the heart, and when subcutaneous, or even when more truly intra-thoracic, it may in these respects efficiently enough simulate an aneurysm; but the pulsation, though expansile from being communicated through fluid, is always less forcible than that of the heart, and there is also entire absence of thrill or murmur, coupled with the presence of the signs, history, and symptoms significant of an empyema.<sup>2</sup>

I have already narrated a series of cases in which uncovering of the pulmonary artery gave rise to visible

<sup>1</sup> In one case I have found a truly aneurysmal thoracic pulsation not to be isochronous with the heart (*vide* p. 441, 2nd Ed.), but this exceptional case in no wise invalidates the above statement.

<sup>2</sup> *Vide* an interesting paper by Dr. M'Dowall in the *Dublin Quarterly Journal* for March 1844, "On the Diagnosis of Empyema," especially at p. 16, where the diagnosis between pulsating empyema and aneurysm is fully considered.

pulsation over which a systolic murmur was audible, thus simulating an aneurysm, and I then pointed out the diagnosis of the one from the other.<sup>1</sup> Aneurysm is also not infrequently simulated by uncovering of the aorta, which is sometimes quite normal, but more usually bent or twisted upon itself,—the result of deformity of the spine—scoliosis—and distortion of the thorax. I have already given the details of several such cases,<sup>2</sup> so we need not enter upon their history further at present.

The importance of cases such as those in relation to the diagnosis of thoracic aneurysm is sufficiently evident. The points of resemblance are also quite apparent. The points in which they differed were—first, in the entire absence of all the subsidiary phenomena dependent upon pressure on the neighbouring organs, phenomena which are often obscure and sometimes misleading, but to their consideration I shall presently recur; and, secondly, in the fact that the isochronous pulsations are less forcible than those of the heart. That aneurysmal pulsation is usually more forcible than that of the heart is a diagnostic point to which much attention has not hitherto been paid. It first impressed itself on my own attention as a matter of some moment in the diagnosis of aneurysm in connection with those abnormal arterial pulsations to which I have just referred, as well as other pulsations of a less delusive character. On talking the matter over with the late Dr. Henderson, Professor of Pathology in the University of Edinburgh, a distinguished authority on the subject, and himself a sufferer from the disease, he informed me that this forcible character of aneurysmal pulsation had also struck him as an important point in relation to the diagnosis of aneurysm close to the heart, and he thought he had made reference to this in his writings. This reference I have, however, been unable to verify. On the other hand, various writers on the subject,

<sup>1</sup> Lecture VIII. p. 217.

<sup>2</sup> Lecture XV. p. 386.

among whom I may mention Dr. Greene,<sup>1</sup> have referred to it as a remarkable fact without attempting to explain it or to estimate its diagnostic importance. Other writers, however, in narrating cases of indisputable aneurysm, have mentioned that the pulsation of the sac was less forcible than that of the heart. The explanation of this discrepancy lies in the fact that the pulsation is only forcible when it is fluid; if the sac be lined with fibrine, other elements are introduced which neutralise those in force when only fluids are concerned. In substernal aneurysms—those close to the heart—fibrinous coagula are rarely found, and thus the physical conditions upon which this forcible pulsation depends remain in force just in those cases in which it is of most consequence that they ought.<sup>2</sup>

Whenever, therefore, we have a tumour in the chest which pulsates isochronously with the heart, and at least as forcibly, we have certainly to do with an aneurysm, which, if it does not now present any marked subjective symptoms, has probably at an earlier period been the cause of such, pain being that symptom most apt to disappear during the progress of the disease. Other symptoms and signs arising from pressure upon adjacent organs are more constant and more likely to increase than to decrease, if we except the signs of pressure on certain veins, such as the brachial, which may entirely disappear as the aneurysm shifts its position in the course of its growth.

*Pain* is the symptom most commonly complained of, and it is a most important note of warning, yet, as it may arise from neuralgia (rheumatic) of the external coverings, or from angina, it cannot be depended upon as an unequivocal symptom of aneurysm. We accept pain as a valuable note

<sup>1</sup> *Vide* his paper on "The Symptoms and Diagnosis of Thoracic Aneurysm," in the *Dublin Journal of Medical Sciences* (1837), pp. 233 and 236.

<sup>2</sup> Allen Thomson says the pressure within an aneurysm is equal to the extent of its internal surface multiplied into the force of the blood in that part of the artery from which it springs.—*Cyclopædia of Anatomy and Physiology*, vol. i. p. 663.



of warning, and it is only exceptionally that it is anything more. The pain of commencing aneurysm differs from that of angina in that it is more continuous and more lancinating (sharp) in character, neither is it so oppressive and depressing. It also resembles ordinary neuralgia in that it often disappears for months without any very obvious reason. When the aneurysm affects the large vessels at the root of the neck, or the aorta in this situation, pain shoots across the chest as well as up one side of the neck or down one arm, or it may extend in both directions, sometimes shooting down both arms. When the aneurysm affects the descending portion of the thoracic aorta pain is usually referred to one spot on the back where it is constant; but in such cases pain may also shoot up the neck, or down the arms, and may even localise itself in the shoulder joint. In these cases we can always make out—by auscultation, if not by palpation—localised pulsation just to the left of the spine where the pain is complained of, generally we can also discover a circumscribed dulness, and sometimes a murmur; various symptoms also serve to confirm the diagnosis. It is chiefly in incipient substernal aneurysms close to the heart that pain is so isolated and so important a symptom as almost to entitle it to be called “pathognomonic,” as Dr. Greene has put it. Yet careful watching even of such cases will ere long elicit other symptoms more trustworthy and more truly pathognomonic of aneurysm than pain alone, which is really only an important warning, but always a warning with a query attached. Pain in the situations referred to is chiefly produced by pressure on the intercostal nerves, or on those of the brachial plexus. A constricting pain around the lower part of the chest is occasionally caused by pressure on the phrenic nerve, and this may also give rise to attacks of dyspnœa, singultus, or more rarely to complete paralysis of the diaphragm. Pressure on the pneumo-gastric nerve in its earlier stages is often accompanied by vomiting, or by severe dyspepsia accompanied by flatulence, which is often

relieved by gentle rubbing of the tumour; at a later stage it gives rise to disease of the lung.

*Dyspnœa* is a frequent symptom of aneurysm; it arises from compression of the trachea, of one of the bronchi, or a portion of lung, or of one of the recurrent laryngeal nerves. When one of the recurrent laryngeal nerves is affected, it may simply be irritated by pressure, and we have spasm of a vocal cord, or its function may be destroyed with paralysis as the result; in the former case both inspiration and expiration are affected; in the latter, inspiration alone is impaired. The voice in these cases is altered to a *vox anserina* (shrill whistling voice); more rarely there is complete aphonia. Inspection by means of the laryngoscope at once reveals on which side the affected cord lies. The left recurrent winds round the arch of the aorta close to the origin of the left subclavian artery and is the nerve usually involved; the right recurrent passes round the right subclavian artery and is rarely affected by any aneurysm. If, on inspection, the glottis is found to open freely, and the arytenoid cartilages to retain their normal movements, any dyspnœa present is not laryngeal in origin, and probably depends upon pressure on the trachea or on one of the bronchi. When the respiration is diminished equally on both sides of the chest, we are justified in regarding the trachea as the part compressed, and in such a case the transverse portion of the arch is usually at fault, though the trachea may, in exceptional cases, be compressed by aneurysms either of the ascending or of the descending aorta. On the other hand, compression of a bronchus gives rise to diminution of the amount of air in the part to which it is distributed; in the whole lung if it be a main bronchus; in the corresponding part of the lung if it be one of the smaller bronchi. This diminution of air in the lung gives the percussion note a higher pitch and a more tympanitic character over the part affected,<sup>1</sup> and it may give rise either to faintness of the respiratory murmur

<sup>1</sup> Skoda, *Abhandlung über Perkussion und Auskultation* (Wien, 1844), S. 18.

over the corresponding part of the lung, or more rarely to the sound of bronchial respiration,<sup>1</sup> a most remarkable phenomenon when associated, as in these cases, with a clear tympanitic percussion note. Dyspnœa, arising from aneurysmal compression of the trachea, or of a bronchus, is naturally increased by exertion, and is frequently much relieved by posture, as by leaning forward when the pressure is on the trachea, or by leaning to one side when one of the bronchi is compressed. In these circumstances air is heard to enter the lung more freely when the pressure is removed. Change of posture is thus not infrequently of service in determining the exact position of an aneurysm. Dyspnœa may also arise from direct pressure on the lung itself; it is then frequently associated with symptoms simulating phthisis, and to obtain relief from these the patient often seeks advice wholly unconscious of the actual malady from which he suffers. Still more rarely dyspnœa is caused by pressure on one of the pneumo-gastric nerves, which may either give rise to serious and fatal disease of the lung, or of itself may prove fatal by inducing suffocation. Of all the organs within the chest, the lungs are those most frequently compressed by aortic aneurysm, hence a circumscribed dull patch is one of the commonest signs of this affection.

*Cough* is a frequent symptom of thoracic aneurysm, and, as it commonly arises from irritation of the pneumo-gastric or laryngeal nerves, it usually presents peculiarities which, if not exactly pathognomonic, are at least sufficiently striking to attract attention. In these cases the cough is often loud and barking, and accompanied by a metallic clang. Such a cough is very distressing both to the patient himself and also to the bystanders, and appears far in excess of the necessities of the case, being accompanied, at first at all events, by the expectoration of only a small quantity of

<sup>1</sup> *Vide* a case by Dr. Halliday Douglas in the *Edinburgh Medical Journal* (December 1869), p. 550, also Dr. Greene, *loc. cit.* p. 233. The explanation of this phenomenon is supplied by Skoda, *op. cit.* S. 108.

glairy and frothy mucus. From the persistent irritation of the cough the expectoration by and by becomes copious and muco-purulent. When the cough arises from direct pressure on the lung, the expectoration is copious and muco-purulent very early in the history of the case. At times the expectoration may be rusty or even red, and though this admixture of blood often comes from the lung alone, yet one cannot help being also anxious as to the state of the aneurysm. In what is called a weeping aneurysm the cough is not always severe nor the expectoration copious, but it always contains some fluid blood.

*Dysphagia* is not an uncommon symptom in connection with thoracic aneurysm; it varies from a mere slight difficulty of swallowing, apparently arising from some interference with the œsophageal innervation, to that more complete form of dysphagia dependent upon compression of the œsophagus, in which the swallowing of solids is either impossible or attended with great difficulty. The diagnosis between dysphagia from the pressure of an aneurysm and that arising from organic stricture is seldom difficult, as organic dysphagia is unvarying and persistent, while aneurysmal dysphagia varies from time to time—is complete at one period of the day and wholly absent at another. Aneurysmal dysphagia is also increased by any excitement of the circulation, and it is markedly relieved by position; thus a patient so afflicted may be able to swallow quite well when he frees his œsophagus from pressure by leaning forwards. The careful use of a probang is capable of revealing in such cases, not only pulsation but even a murmur, but this is a proceeding not without risk, or one to be undertaken rashly.

*Pressure on the blood-vessels* produces various well-known signs and symptoms; amongst these we have alterations of the radial pulses, so that one differs from the other in size or volume. To avoid being misled by simple irregular distribution of the radial artery, we must carefully ascertain whether the difference in volume extends to the brachial also. A



sphygmographic tracing from both radial arteries shows at once whether we have to do with simple abnormal distribution or with an abnormal blood-wave. The alteration of the blood-wave is, however, rarely, if ever, due to pressure, but almost invariably to stenosis of the subclavian or brachial artery from *endarteritis deformans*, which not only deforms the pulse but may even delay it. Hence the diagnosis of aneurysm can never be made with certainty from pulse changes alone.<sup>1</sup> Pressure on the brachial veins produces swelling of the corresponding arm, unaccompanied by any signs of inflammation. The dependence of this swelling upon aneurysmal pressure is made probable when it comes on suddenly during violent exertion, and especially if there is an abnormal pulsation or murmur localised in the innominate, subclavian, or carotid arteries. Should the aneurysm shift its position and develop in some other direction, this swelling may disappear and a pulsation may then appear about the upper part of the chest or the root of the neck. Considerable compression or even obliteration of the superior *vena cava* has been occasionally observed, attended by great development of the superficial veins of the arms, neck, and face. When the compression is not so great, a moderate development of the superficial veins is not infrequently observed; and in some of these cases this takes the form of a thick œdematous collar covered with enlarged veins surrounding the root of the neck. The interference with the cerebral circulation thus produced gives rise to various head symptoms, headache, quasi-apoplectic seizures, etc. Compression of the *vena cava descendens* or of the right auricle may give rise to congestion of the abdominal organs and dropsy, but these are usually late symptoms, and there are then plenty of other symptoms and signs present to lead us to the true diagnosis.

*Pressure on the heart* by aneurysm is not an uncommon cause of displacement of that organ. If the aneurysm

<sup>1</sup> *Die Untersuchung des Pulses*, von Dr. M. v. Frey (Berlin, 1892), S. 238.

implicates the ascending aorta, the heart is displaced downwards and to the left; if the transverse part of the arch is affected the displacement may be downwards solely; and when the descending aorta is affected, the heart may be displaced to the right, or if the aneurysm is located just behind the heart, that organ is compressed against the anterior wall of the thorax and gives a larger and more forcible impulse. Consecutive alterations of the heart connected with aneurysm are rarely of any importance; even when a double murmur at the base of the heart reveals incompetence of the aortic valve, a condition which in ordinary circumstances is attended by great consecutive changes, these, when the incompetence is caused by aneurysm, are generally so slight as to be unimportant, and have little bearing on the ultimate progress of the case.

At times the pressure of an aneurysm is so great as to depress the liver and to communicate to it well-marked pulsation; of this I have seen one very interesting example (*vide* p. 8).

When the pressure of an aneurysm affects the *nerves issuing from the spinal cord* in what is called "the cilio-spinal region," that is, according to Budge and Waller, from the sixth cervical to the sixth dorsal nerve, or, according to Brown-Sequard, as low as the ninth or tenth dorsal nerve, certain phenomena are observed which vary with the amount of pressure exerted. From the anterior roots of the nerves referred to certain filaments pass through the cervical sympathetic to the iris and are distributed to the *dilatator pupillæ*, consequently when the aneurysmal pressure in the region referred to is but slight these nerves are only irritated and the pupil is dilated. When the pressure is considerable the nerves are paralysed and the pupil is contracted. When contraction is extreme the effect is very striking, but there are a multitude of cases in which the difference between the two pupils is but trifling, and in which it is difficult to decide which pupil is abnormal. Further, we must remember that

an average of one person in fourteen has one pupil smaller than the other. When we find the pupils of the two eyes differing in size we must first of all carefully measure both, and then proceed to test them. The contracted pupil of spinal myosis is insensible to light and does not further contract on exposure, but it does contract on accommodation for near objects; further, the reaction to atropine of such pupils is always incomplete. When the left pupil is thus ascertained to be affected with spinal myosis we know we have to do with some affection of the cilio-spinal nerves, the exact nature of which has yet to be discovered, but which may be an aneurysm. Spinal myosis on the right side has no connection with aortic aneurysm. Even on the left side spinal myosis is not pathognomonic of aneurysm, but it may help us to detect one by directing special attention to the region involved.<sup>1</sup>

*Circumscribed dulness* is always present when the aneurysm reaches the wall of the thorax. From the more or less globular shape of an aneurysm the tumour is invariably larger than the area of complete dulness. Over this dull patch we always have pulsation which is more or less fluid and forcible in character, according to the thinness of the walls of the sac and the amount of clot contained within it. The most common situation of aneurysm, where, therefore, this dull patch is most usually found, is on the right side of the sternum, on a level with the second or third ribs; more rarely it is to be found on the left side of the sternum, or over that bone itself, or at the back. In the latter case the aneurysm usually arises from the posterior part of the descending aorta, and it only rarely passes to the right of the spinal column. As the disease progresses the circumscribed dulness increases; the thoracic wall becomes involved in the

<sup>1</sup> *Vide* Dr. Gairdner in the *Edinburgh Medical Journal* for January and August 1855; Dr. Ogle in the *Medico-Chirurgical Transactions*, vol. xli.; and for the physiology of the subject two interesting papers by Dr. Argyll Robertson in the *Edinburgh Medical Journal* for February and December 1869.

tumour, which finally breaks through and appears on the surface as a pulsating swelling; in this condition its true character is not likely to be mistaken.

Over the dull pulsating tumour various *sounds* are to be heard, and these vary in each case. Perhaps what we call a murmur is the rarest of all sounds heard over a thoracic aneurysm, a systolic jog being all that is appreciable in many cases, though occasionally this jog is double. In these cases it appears probable from the firmness of the impulse that the sac is well coated with fibrine internally, but this is certainly not always the case. Most commonly the sounds audible over an aneurysm are merely the normal sounds of the heart, and when this is the case it has long been known, having been first pointed out by Dr. Henderson in 1836, that the second sound, that produced by closure of the aortic valve, presents what he has called "a very striking resemblance to the shutting of a pump-valve in the immediate vicinity of the ear,"<sup>1</sup> and this has been variously designated by succeeding observers as a ringing, booming, or accentuated second sound. This accentuated or booming sound, when heard over a circumscribed dull patch, and limited to that position, is very distinctive of aneurysm. If, however, this booming sound is produced so near as to be readily propagated into the aortic area, it is apt to be confounded with a similar accentuation due to dilatation of the ascending aorta.<sup>2</sup> Hence an accentuated second heard over a circumscribed dull patch out of the aortic area is almost distinctive of aneurysm; heard within that area alone it indicates dilatation of the ascending aorta; but if it is also propagated to the right or left of the normal course of the aorta, the probabilities are in favour of the dilatation being combined with bulging (cirroid aneurysm), rather than that there is any true saccular aneurysm. When a double murmur is to be heard at the base of the heart, the same murmurs, only intensified,

<sup>1</sup> *Edinburgh Medical and Surgical Journal*, vol. xlv. p. 316.

<sup>2</sup> Lecture I. p. 31.



are also heard over the aneurysm. At times a systolic murmur of varying character is heard over the aneurysm preceding the accentuated second sound, and still more rarely this systolic murmur is followed by a loud diastolic murmur. In the only case in which this occurred in my own experience, the opening of the aneurysm was large and smooth, and it was difficult to account for the occurrence of a diastolic murmur at all (*vide* p. 441, 2nd Ed.) A localised murmur heard anywhere in the course of the aorta, or of its larger branches, is always a suspicious sign, and ought to lead to further inquiry. But inasmuch as a murmur of this character may arise from a projecting spicula, or from the pressure of a tumour, it is of little value by itself and is only conclusive when associated with other confirmatory signs and symptoms.

*Undue delay of the pulse* is a sign of endarteritis, atheroma, or dilatation of the aorta, but not of saccular aneurysm, which does not interfere with the normal propagation of the pulse.

A knowledge of the anatomical relations of the thoracic aorta teaches us that the predominance of one or other of the symptoms just described may be accepted as a certain indication of the site of an aneurysm, and often of its size. *Pain* on the right side is of comparatively little consequence, as an aneurysm coming from the ascending aorta and passing to the right lies in front of everything, and has already involved the anterior chest wall before pain becomes a prominent symptom. On the left side it is different, and so-called rheumatic pains about the left shoulder ought always to be most carefully inquired into, as not infrequently they are the earliest indication of an aneurysm of the upper part of the descending aorta. If an aneurysm in this situation is of any size we frequently have either dilatation or contraction of the left pupil; but this is an inconstant sign and never an early one; it is therefore of less importance than pain. *Brassy, spasmodic cough*, and *vox anserina*, lead us at once to the left edge of the sternum about the first interspace, just over the

left end of the transverse part of the arch of the aorta where the left recurrent nerve winds round it. *Dyspnœa*, not accompanied with brassy cough, or vox anserina, and not associated with any obvious affection of the lungs or heart, should lead us to examine the region beneath the upper part of the sternum with great care, as it will not infrequently be found to depend upon pressure upon the trachea or the right bronchus by an aneurysmal bulging of the first half of the transverse portion of the arch of the aorta. In rare cases this bulging is strictly limited to the posterior part of the vessel, and a positive diagnosis is quite impossible, yet the signs and symptoms are even in these cases so well marked and so distinctive as to make the probability almost a certainty.<sup>1</sup> Aneurysms in the posterior mediastinum present the fewest symptoms of any, and are seldom even surmised until they produce pain by erosion of the vertebræ, or have become large enough to implicate the lung, or to interfere with the circulation through the *azygos*, *hemiazygos*, or *vena cava ascendens*. When an aneurysm springs from the anterior part of the ascending aorta, it occasionally passes to the left, and, getting in front of the lung, just above and in close proximity to the heart, it sometimes escapes observation until by pressure it has produced considerable disorganisation of the lung.

The diagnosis of abdominal aneurysm is either very easy or the reverse. During a considerable part of its course the abdominal aorta can be readily felt, and any alteration of its calibre distinguished by palpation. Should any pulsating tumour be detected, auscultation will usually reveal a systolic murmur solely; the second sound is not propagated into the abdominal aorta, and a diastolic murmur is almost unknown.<sup>2</sup>

<sup>1</sup> The case of the late Earl St. Maur will at once recur to the reader, and at p. 1123 of the *Edinburgh Medical Journal* for June 1872 will be found a similar case, in which an equally successful attempt at diagnosis was made.

<sup>2</sup> The only recorded case of diastolic murmur in abdominal aneurysm, that I am acquainted with, is one narrated by Dr. Wickham Legg in *St. Bartholomew's Hospital Reports* for 1880, vol. xxi. p. 258. I myself have also met with one similar case.

When the aneurysm springs from the anterior surface of the abdominal aorta, no pain is complained of unless it arise very high up in its course, and almost if not quite beyond the reach of palpation. Occasionally we have flatulence and other symptoms of gastric disturbance, apparently the result of interference with the innervation of the stomach. The immobility of a tumour is no proof of its aneurysmal character, as Dr. Henry Kennedy supposed; it only shows that it is fixed, it may be, by adhesions or otherwise, but it is not any proof that it springs from the aorta. When an aneurysm arises from the posterior surface of the aorta, and especially if it be beyond the reach of palpation, it is chiefly *per viam exclusionis* that we arrive at our nearest approximation to the truth. Perhaps the most distinctive symptom is an otherwise unaccountable neuralgic pain affecting the bowels, passing as a girdle pain round the body, or shooting down the spine into the extremities. This pain is tolerably persistent, but is subject to variations like all other symptoms of aneurysm, and now and then it disappears for months at a time; indeed this unaccountable disappearance may be regarded as to a certain extent confirmatory of the diagnosis of aneurysm. Dr. Beatty of Dublin has related a most instructive case of abdominal aneurysm, in which the tumour lay between the *crura* of the diaphragm, and in which the character of the pain led ultimately to what proved to be a correct diagnosis.<sup>1</sup> It is to be regretted that neither percussion nor auscultation down the spine were resorted to in this instance; they ought never to be omitted, as it is quite unusual to obtain no information from these methods of exploration, and the discovery of a localised dulness or an arterial whiz materially aids the diagnosis in these cases.

The course of an aneurysm is usually chronic; it may last for years, till death takes place either gradually from asthenia or dropsy, or more suddenly from cedema, gangrene, or inflammation of the lungs, or from rupture of the sac, either

<sup>1</sup> *Dublin Hospital Reports*, vol. v. p. 166.

externally, or into one of the serous cavities, or into a mucous canal. Death from pure asthenia is comparatively rare, a gradually increasing marasmus being usually hastened to its fatal termination by some acute complication or intercurrent disease, or by rupture of the sac. Rupture of the sac is not always immediately fatal; rupture into a serous cavity usually is so, but there are many cases of so-called "weeping aneurysms" in which the sac communicates with some mucous surface, and pours forth small quantities of blood at irregular intervals for months or years. When this leakage is in connection with the alimentary tract it is not so obvious, except when so copious as to give rise to hæmatemesis, a state of matters but rarely followed by recovery, and yet there is at least one case of this upon record.<sup>1</sup> When the blood is poured into the respiratory tract attention is at once attracted to this alarming symptom. In Mr. Liston's case the period that elapsed between his first copious hæmoptysis and his fatal hæmorrhage was five months, without any intervening bleeding. Dr. Gairdner has related a remarkable case in which, from the first leakage to the last fatal effusion, no less than five years elapsed, during which there were repeated slight attacks of hæmorrhage.<sup>2</sup> But the most remarkable cases of intermittent hæmorrhage are those recorded as having taken place from the external surface. Thus Neligan relates the case of a ship carpenter, with an aneurysm of the aorta opening externally about the second rib on the right side in front, which for more than a year discharged blood at intervals, and that sometimes so copiously and in so full a stream as to be with difficulty arrested. Three weeks after his last bleeding his aneurysm underwent a marked abatement, and he left the hospital declaring himself to be quite well.<sup>3</sup> Nor is this case unique; several somewhat similar cases have been recorded. Perhaps the most extraordinary of these is

<sup>1</sup> Gairdner's *Clinical Medicine* (Edinburgh, 1862), p. 495.

<sup>2</sup> *Op. cit.* p. 509.

<sup>3</sup> Stokes' *Diseases of the Heart and Aorta* (Dublin, 1854), p. 582.



one narrated to the late Mr. Syme, by Mr. Ramsay of Broughty-Ferry, of a man with aneurysm of the arch of the aorta and of the innominate artery which ruptured opposite the cartilage of the third rib. A stream of blood somewhat thicker than a quill is reported to have issued from the opening, the patient, nowise alarmed, held a bowl to receive the contents of what he supposed to be a "bloody boil," and even squeezed it with his chin to empty it faster; when he had lost about a quart of blood he fainted, and the bleeding ceased. Four months subsequently he died of typhus, the bleeding having never recurred.<sup>1</sup>

Thus even rupture of an aneurysm, usually and rightly regarded as almost necessarily fatal, does not always make further treatment utterly hopeless; and if the case be so with a ruptured aneurysm, with how much more hope ought we not to attempt the cure of one still unruptured. Unaided nature has not infrequently succeeded in promoting the cure of such cases; and the medical art would be unworthy the confidence reposed in it were it incapable of following up and improving upon the hints afforded by nature as to the treatment of this most serious disease. For nature can only cure in certain conditions and in certain circumstances, but art can change the latter and modify or fulfil the former. Art, therefore, properly directed, ought to cure more than nature; and as no case is utterly hopeless even when left to nature alone, so all ought to be more or less hopeful when under the guidance of art; and the more hopeful the more intelligent the art that guides them is.

The spontaneous or natural cure of aneurysm is effected in three modes—First by sphacelus of the tumour, the arterial opening remaining closed; second, by occlusion of the artery from which the aneurysm springs; and, thirdly, by gradual occlusion of the aneurysmal sac with fibrine, the artery from which it springs remaining pervious.<sup>2</sup> The first

<sup>1</sup> *Monthly Medical Journal* (January 1850), p. 89.

<sup>2</sup> *Vide* Hodgson *On Aneurysm* (London, 1815), p. 101. Many cases of spontaneous cure of aneurysm are to be found in this work.

method, sphacelus, can scarcely be called a method of cure, though in rare instances it has accidentally been followed by this result; it certainly cannot be imitated by art. The second method is that upon which the surgical treatment of external aneurysms is based; it is obviously inapplicable to the treatment of internal aneurysms connected with the main trunks of arterial supply. The medical treatment of internal aneurysms has therefore been largely restricted to an endeavour to bring about the third mode of cure—occlusion of the sac by fibrine. This method is of course inapplicable to



FIG. 32.

cirroid aneurysm (those with bulgings only). Happily there is a fourth method of treatment thoroughly scientific in its aims and applicable to all forms of internal aneurysms, but consideration of this I shall reserve for a future chapter.

Of the spontaneous cure of aneurysms of the aorta, I have only seen two examples. The first was a small aneurysm, rather larger than a large walnut, wholly filled with a firm, pale, fibrinous clot, and springing from the upper part of the ascending aorta just where it passes into the transverse part of the arch. The specimen (Fig. 32, *a* is placed on the aneurysm) was removed from the body of a man who died from the fatty form of Bright's disease (large white kidney),

and was never known to have exhibited any symptoms of aneurysm.<sup>1</sup> The second case was that of an aged woman, an inmate of one of our eleemosynary hospitals in charge of the late Dr. Gillespie. In this case the aneurysm sprung from the descending part of the aorta, just after leaving the transverse part of the arch (Fig. 33). The aneurysm passed backwards into the left vertebral sulcus, and pressed upon the left lung. Hæmorrhage from this lung, unconnected with the artery but caused by disorganisation induced by pressure, proved fatal. The aneurysm was as large as an orange, was wholly filled

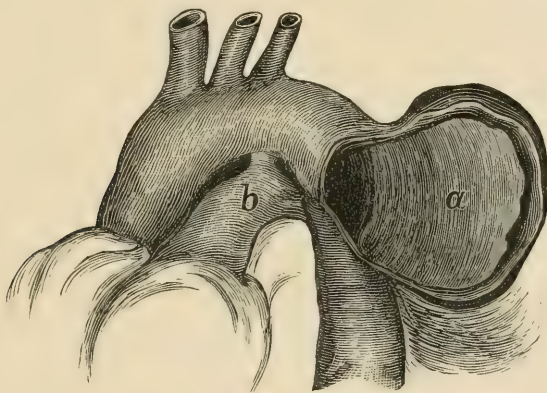


FIG. 33

with a laminated clot, and had presented none of the usual symptoms; it had in fact been wholly unsuspected. In this case the whole of the arch had been converted into bone (atheroma), and was as firm as the handle of a walking-stick.<sup>2</sup>

Attempts have been made in various ways to produce this favourable termination. Every variety of sedative has been employed with this hope, but so unsuccessfully as to require no notice here. Even their use as palliatives has been of little avail, and the most powerful anodynes have been almost

<sup>1</sup> *Edinburgh Medical Journal* (July 1870), p. 82.

<sup>2</sup> Both of these preparations are in the Museum of the Royal College of Surgeons, Edinburgh.

useless in relieving the excruciating agony that so commonly accompanies this terrible disease.

Of all the more modern attempts to cure or relieve this affection there are six which seem worthy of mention, and the first of these I shall notice only because it is modern, and not because its employment has been either markedly beneficial or deserving of imitation. Perhaps there is nothing that testifies so strongly to the dangerous character of aneurysm, and to the inefficacy of the remedies hitherto employed, than the fact that learned physicians have been found to advocate the *introduction of fine iron wire* into an aneurysm with the view of providing an extensive surface upon which the fibrine might coagulate. The late Dr. Murchison and Mr. Charles H. Moore introduced twenty-six yards of such wire through a fine pointed cannula into a saccular aneurysm of the ascending aorta. The treatment was unsuccessful; it could scarcely have been otherwise, as it was not employed till it was clear that the patient had not many days to live. The proposers of this treatment contend that their experiment showed that the principle upon which it was based is sound, and that further experiments are justifiable.<sup>1</sup> But surely nothing could justify so hazardous an experiment except the full conviction that no less dangerous treatment is available, and to this, of course, I demur, as I think I shall be able to show that there is a treatment wholly free from danger which holds out sufficient prospect of success to preclude our having recourse to any such hazardous experiments. Besides, this treatment is only applicable to saccular aneurysms, and it is not always easy to ascertain whether an aneurysm is truly saccular or not. If this condition could be distinctly recognised there are few, I think, who would not prefer the less dangerous and more efficient agency of *electrolysis*. Unfortunately we can never be perfectly certain that an internal aneurysm is truly saccular, and in any other the risk of embolism, though probably exaggerated, is yet so great as to

<sup>1</sup> *Medico-Chirurgical Transactions*, vol. xlvii. p. 129 (London, 1864).



prevent the use of galvano-puncture in all but exceptional cases, and its employment is chiefly restricted to those external arterio-venous tumours which are known as surgical "cirroid aneurysms," in the treatment of which it is most successful. Provided always that the arterial element prevails; when the venous element is in excess I can say with perfect certainty that galvano-puncture is of no use if the disease is of any extent. In truly medical aneurysms the chief use of galvano-puncture is to prevent external rupture, and for this, other measures having failed, it is admirably adapted, and is therefore deserving of a short notice. Galvano-puncture was originally suggested by Pravaz for the cure of external aneurysms, in the treatment of which it has had a fair measure of success, as may be seen by reference to Ciniselli's memoir,<sup>1</sup> to which I need not now further refer. Its employment has been extended to thoracic and abdominal aneurysms by Ciniselli,<sup>2</sup> by Drs. James and John Duncan,<sup>3</sup> and by Dr. Decristoforis,<sup>4</sup> who have narrated eight cases of thoracic aneurysm, in only one of which was any considerable degree of success attained;<sup>5</sup> the others were all unsuccessful. In one case of abdominal aneurysm complete success seemed to have been attained, but unfortunately premature exertion was followed by the sudden death of the patient.<sup>6</sup> The results attained in the treatment of internal aneurysm have not thus been such as to lead us to hope much from the employment of galvano-puncture as a remedial agent in this class of cases, while the unavoidable risks of embolism are so great as to lead to its abandonment by almost all. On the other hand, the ease and rapidity with which clots can be formed by means of the galvanic current must certainly lead

<sup>1</sup> *Sulla elettro-puntura nella cura degli aneurysmi* (Cremona, 1856); and *Edinburgh Medical Journal* (April 1866), p. 926.

<sup>2</sup> *Edinburgh Medical Journal* (April 1866), p. 926.

<sup>3</sup> *Ibid.* p. 920; and August 1867, p. 101.

<sup>4</sup> *Aneurysm dell' aorta ascendente trattato coll' elettro-puntura, caso clinico* (Milan, 1870), etc.; and *Edinburgh Medical Journal* (December 1870), p. 537.

<sup>5</sup> *Edinburgh Medical Journal* (December 1870), p. 540.

<sup>6</sup> *Ibid.* (April 1866), p. 922.

to its employment, as a *dernier ressort*, in all cases where external rupture seems imminent. For galvano-puncture of aneurysms steel needles are employed, about five inches in length, and of the thickness of 0·07 of an inch, that is No. 16 of the wire-gauge. The non-insulated portion of each needle should measure one inch and one-eighth of an inch in length, while the insulated portion, or that coated with vulcanite (or ebonite) should measure three inches and three-eighths. If the steel be not gilded the positive pole is rapidly corroded; but when the wire is of the thickness prescribed, it is not acted upon to such an extent as to have its efficiency seriously impaired. Moreover, when blood is the electrolyte, the corrosion of the steel results in the production of ferrous chloride and probably also of ferrous sulphate, salts which coagulate albumin, and thereby the wished-for result is hastened.<sup>1</sup> Four cells of a Bunsen's battery are sufficiently effective and give rise to no pain, or to only a bearable amount of pain. But as even six cells sometimes produce so much pain as to make the patient uncontrollable,<sup>2</sup> if it is thought desirable to use more than four cells, the patient should be put under chloroform.

The battery should have a continuous current, and the needles should be introduced from the same side, near the base of the external tumour, parallel to each other, and one or two inches apart. The number of needles may be multiplied if the aneurysm is large, and the action may be prolonged till pulsation ceases, or till gas can be clearly detected on percussion. A *séance* of about twenty minutes is generally enough for one application, and this may be repeated as necessary according to the circumstances of the case. Such is a succinct account of the method of employing galvano-puncture in aneurysm—a method of treatment that cannot be recommended for the cure of internal aneurysms, both because of the risks connected with it and also because we

<sup>1</sup> *Edinburgh Medical Journal* (August 1867), p. 119.

<sup>2</sup> *Ibid.* p. 109.

have a safer and a better method ; but it is a treatment which may be very advantageously employed as a means of prolonging life in exceptional circumstances. Last winter (1874-5) we had a very instructive case in which we had unavailing recourse to galvano-puncture.

CASE XL. Alexander McIntosh, a mason, aged thirty-four, residing at Newport, Fife, admitted to Bed 10, Ward V., on 12th October 1874, complaining of a pulsating tumour at the root of the neck, and another actually beneath the right shoulder-blade. He is a hardworking man of temperate habits, with a comfortable house, and a good family history, who had met with no more serious accident than a fall on his right elbow joint about six months previous to his admission. About six weeks before admission, while at work, he was seized with a pain between his shoulders ; in itself this was of no consequence and did not prevent his continuing at work, but it directed his attention to the spot, and there he discovered a swelling. On consulting his doctor he was told to stop work and apply for admission to the Royal Infirmary. He has now no pain, only a feeling of stiffness over the right shoulder and upper part of the chest. With the exception of an attack of fever, about ten years ago, which laid him up for about two months, he has always had good health. He is a well-developed man in good condition ; weight, 11 stone 4 lb. ; height, 5 feet 7 inches ; skin natural ; temperature 98·4. For the last six weeks he has suffered occasionally from palpitation ; he has a feeling of stiffness about the upper part of the chest in front. Both radial pulses are synchronous and of nearly equal force, though at times the right seems the feebler ; the pulse is weak, 90 per minute. Above the right clavicle there is a bluish lobulated swelling, which commences a little to the right of the middle line and runs round the neck to the back ; about the middle of the clavicle it measures three inches across. Posteriorly in the region of the right scapula there is a visible pulsation, the margins of which are ill-defined.

Beneath, the pulsation extends a little beyond the scapular angle; internally, where it is most prominent, it almost reaches the middle line; externally, it reaches the posterior fold of the axilla; and above, it rises to the level of the *vertebra prominens*, and merges in the swelling surrounding the neck. Posteriorly the pulsating tumour does not differ in colour from the natural skin. On palpation the swelling over the clavicle feels soft, fluid, and relaxed, except at one spot just above the clavicle, where a firm and more elastic tumour is to be felt. On placing the hand lightly over this soft swelling a purring tremor is felt, but no distinct pulsation until we press deep enough to reach the elastic tumour just alluded to, which feels rounded and beats a little later than the cardiac apex. The pulsation behind is heaving, and lifts the scapula at each beat; at its upper part there is a purring thrill similar to what is felt in front, but less intense. Firm pressure on the elastic tumour referred to as projecting above the clavicle stops the thrill both in front and behind, and greatly diminishes the size of the tumour behind as well as the force of its pulsation. The cardiac apex beats in the fifth interspace, three inches from the middle line; the cardiac pulsation seems to occupy a larger space than usual, but is not forcible; there is no pulsation in the episternal notch, but there is considerable heaving over the upper part of the right side of the chest. The cardiac dulness does not rise above the third rib; at the level of the fourth costal cartilage it commences at the right margin of the sternum and extends to the left for a distance of five inches. On the right side anteriorly the upper part of the chest is dull down to the third rib; as low as the first interspace this dulness reaches the middle line; beneath that there is a space of about two inches to the right of the middle line where the percussion note is more resonant than over any other part of the chest. Posteriorly the whole right half of the chest is dull down to the lower angle of the scapula; beneath this there is normal resonance for the space of about two



inches, gradually shading into complete liver dulness. The percussion on the left side is normal, both in front and behind.

On auscultating over the heart's apex the first sound is found to be impure; the second unusually loud. Over the upper part of the sternum and the dull part to its right, there is a rough double murmur audible as low down as the fourth costal cartilage; at this spot, however, there is only a systolic murmur to be heard followed by a second sound. In the pulmonary area the second sound is obscured by the diastolic murmur, but it may be picked up quite pure on passing the stethoscope to the left in the plane of the second interspace. The rough double murmur continues audible over the innominate, but over the rounded pulsating tumour above the clavicle only a loud systolic murmur is to be heard. Over the bluish, lobulated, venous tumour, especially close to the trachea, a loud purring whiz is audible, and this is so loudly propagated into both carotids as entirely to obscure any other acoustic phenomena which might perchance have been audible in them. Over the upper part of the posterior tumour there is a loud whiz; over its lower part merely a systolic murmur. Over the left lung the respiration is vesicular, but somewhat feeble. On the right side the respiratory sounds are entirely obscured by the murmurs described, except in the part beneath the scapula, where the respiration is vesicular but slightly higher in pitch than on the left side, and a similar statement may be made as to the respiratory sounds heard over the lower part of the right lung anteriorly. The patient's other organs and systems are normal.

Here we had to do apparently with an aneurysm of the ascending part of the aorta, involving the innominate and subclavian, with a bulging projection from the latter vessel which communicated with the veins of the neck, its history and mode of origin being obscure. It was impossible to decide as to the character of the posterior pulsating tumour.

The patient was seen and examined by almost all of the physicians and surgeons attached to the Infirmary, but no definite opinion was arrived at. It seemed to be either an aneurysmal dilatation of the posterior scapular artery, or a cirroid aneurysm involving it and its branches. There was no reason to suppose that it arose from the aorta perforating the chest posteriorly. Under either of the suppositions suggested it seemed to be a favourable case for galvanopuncture, and this all the more that the pulsation could be almost completely controlled by pressure above the clavicle. My colleague, Dr. John Duncan, whose large experience in regard to galvano-puncture is so well known, was kind enough to undertake the treatment under my supervision, and continued it for more than six months. After each application of the needles the circulation in the tumour was controlled and the pulsation suppressed by a large bag of shot firmly bandaged over the supra-clavicular space. After each application the usual results followed, the development of a certain amount of gas and the consequent diminution of the pulsation, but it was several months before any firm nodular coagula could be detected. At last, however, these made their appearance and gradually spread, till we hoped that a cure of this part of this poor man's ailment was about to be secured. This hope was delusive; these coagula never accumulated or coalesced; on the contrary what seemed to be gained at one *séance* was too often lost before the next, though they were not unduly postponed, but were repeated at as short intervals as possible. At last the patient was discharged on 8th May somewhat improved, and the pulsation beneath the scapula lessened. I have seen him since his discharge in his own house at Newport and found him much as he was on admission, all that seemed to be gained having been lost. I have not seen him lately, but have just (1897) heard from his medical attendant, Dr. Stewart, that M'Intosh is in excellent health and has been working at his trade for some years without discomfort. If we cannot claim M'Intosh

as a cure, he has certainly lived twenty years longer than many so-called cures.

Professor MacEwen of Glasgow started the idea that galvano-puncture coagula were apt to melt away because they were merely red clots, and that in the cure of aneurysm the only clots of any value were the white *thrombi* originating at the point of injury to the sac wall as part of a reparatory inflammatory process. With this idea he introduced a needle into the sac, and by scratching the lining wall he started quite a number of reparatory *foci* with their white *thrombi*. MacEwen treated four cases of aneurysm in this way, and claims that he cured two of them.<sup>1</sup>

Dr. Stewart of Philadelphia has, within the last few years, introduced a combination of the wire treatment with galvano-puncture which seems to combine the advantages of both methods.<sup>2</sup> Stewart introduces ten feet of snarled, coiled, fine gold wire into the sac and passes a galvanic current through this, with the result of rapidly filling the sac with firm coagula. Three to five feet of wire is sufficient for a sac three inches in diameter; this is introduced through a needle, and attached to the anode or positive pole, which should invariably be the active electrode. The negative rheophore should be a large clay plate, or an absorbent cotton pad made after the method of Massey, and should be placed upon the abdomen or the back. The current should be slowly brought into circuit and its strength gradually noted by an accurate milliampère-meter. The strength of the current should be rather high—from forty to eighty milliampères, and the sitting long—from three-quarters of an hour to an hour and a half. All areas of the sac are thus reached through the coiled and snarled wire, and consolidation of the contents by means of clot formation is promptly and invariably produced. Dr. Stewart has given details of several cases treated by himself and others in this manner, which is certainly the most

<sup>1</sup> *British Medical Journal* (November 1890), pp. 1107 and 1164.

<sup>2</sup> *Ibid.* (14th August 1897), p. 387.

scientific and apparently the most hopeful method of employing galvano-puncture.<sup>1</sup>

The late Professor Langenbeck of Berlin<sup>2</sup> strongly recommended the *hypodermic injection of ergotine* as a cure for aneurysm, on the strength of two cases which were thus successfully treated by him. The first was an aneurysm of the subclavian and innominate arteries, in a man aged forty-five. He had a pulsatile swelling the size of one's fist in the right supra-clavicular fossa, with excruciating pain in the right arm, and consequent sleeplessness. From 6th January to 17th February 1869 injections of ergotine of from half a grain to three grains were made at regular intervals of about three days. This treatment was followed by great relief to the patient and considerable diminution of the tumour. The second case was a saccular aneurysm of the radial artery, in a man aged forty-two. One single injection of ergotine was followed by complete subsidence of the tumour. Next day the aneurysm had in some degree returned, and there was slight swelling and infiltration in the neighbourhood of the puncture. After eight days these symptoms had all disappeared, and not a trace of the tumour could be perceived. Langenbeck's accuracy and skill as an observer were too well known to permit his observations to be ignored; at the same time, if the action of ergotine be, as he supposed, on the unstripped muscular fibre, it is difficult to see what possible result this could have had on the larger tumour described, as the muscular fibres surrounding it must have been few and sparse indeed. It seems possible that this treatment may have some effect on small aneurysms connected with small arteries; but for the reason stated it seems impossible that it could have any effect on large aneurysms, especially those springing from large arteries. I have employed the hypodermic injection of ergotine repeatedly, and in one case

<sup>1</sup> Vide *American Journal of the Medical Sciences*, October 1892 and August 1896.

<sup>2</sup> *Berliner klinische Wochenschrift*, March 1869, and *Edinburgh Medical Journal* (November 1869), p. 461.



continuously for many months, without observing the very slightest result, and yet the preparation was unquestionably active, as proved by the excellent results we obtained by its use in hæmorrhages, both in hæmoptysis and in epistaxis. It would, however, be unfair to omit all allusion to this subject in any treatise purporting to present modern views of the treatment of aneurysm. The preparation used by Langenbeck was Bonjean's watery extract diluted with three times as much rectified spirit and glycerine. That which I employed was prepared in this country, and was undoubtedly active, as proved therapeutically as well as physiologically, one grain of the preparation hypodermically injected sufficing to stop completely the circulation in a frog for fully five minutes.<sup>1</sup>

*Pressure*, as a mode of treating aneurysm, however useful in regard to external aneurysms, is of course wholly inapplicable to those within the thorax, and also as a rule to those within the abdomen. One case of abdominal aneurysm has, however, been recorded by Dr. William Murray of Newcastle-on-Tyne, in which pressure on the aorta immediately above the tumour was successful in curing the disease. The first attempt in this case, a man aged twenty-six, failed. On the second attempt the patient was kept for five hours under chloroform, and pressure was fully maintained by a properly constructed tourniquet. The result was that in three months the patient was at his work as an engine-fitter, perfectly cured. The tumour itself, the aorta below it, the iliac and femoral arteries were quite pulseless.<sup>2</sup> Cases of abdominal aneurysms in which this treatment is available must be rare, but it may be more frequently useful in iliac aneurysms, and it is well to remember that, in fitting cases,

<sup>1</sup> *Vide* a paper by the late Dr. Hirschfeld, "On the Action of Ergot and Ergotine," in the *Medical Press and Circular* (February 1870), p. 139.

<sup>2</sup> *Medico-Chirurgical Transactions*, vol. xlvii. p. 187 (London, 1864); and *Medical Times and Gazette* (April 1865), p. 383, in which is a further report of the case, showing the cure to have been complete; *vide* also the *Rapid Cure of Aneurysm*, by William Murray, M.D., F.R.C.P. (London: J. and A. Churchill, 1871).

we have in pressure a remedy capable of producing a much-to-be-desired result in a comparatively short time. The treatment which, however, has for long been deemed by physicians as that most suitable for internal aneurysms, is that called by the name of its most strenuous advocate, *Valsalva's treatment*, though, as we learn from Morgagni, Albertini had an almost equal predilection for its use. When pushed as far as its originator desired, this consisted in weakening the patient by repeated blood-lettings, and by gradually diminishing his food and drink till only half a pound of pudding was taken morning and evening, along with a measured quantity of water.<sup>1</sup> This was continued until the patient was so reduced that he could not lift his hand from the bed in which, by Valsalva's orders, he lay from the commencement of the treatment. After this the quantity of nutriment was gradually increased till the patient's strength was again restored. We have the testimony of many physicians that this treatment has been useful in many cases, and it can scarcely be thought necessary to adduce *seriatim* evidence in favour of a practice originally suggested by Hippocrates,<sup>2</sup> and which after a varying fashion has kept its place in therapeutics ever since.<sup>3</sup> In recent times the late Dr. Bennett of Edinburgh attempted to revive this treatment. His patient was repeatedly purged freely; he twice had a dozen leeches applied over the tumour; was twice bled to  $\bar{\text{xxii}}$ , and several times to syncope, while his diet was extremely low, his dinner being only  $\bar{\text{iii}}$ . of steak and  $\bar{\text{iii}}$ . of bread, yet after forty days' treatment he retained sufficient strength to walk two hundred and fifty yards to the nearest cab-stand on his way from the city. Bennett expressed his satisfaction with the treatment and his determination that his next case should be even more rigorously treated.<sup>4</sup> But in spite of its occa-

<sup>1</sup> Morgagni, *De Sedibus et Causis Morborum*, Letter xvii. Art. 30.

<sup>2</sup> *De Morbis*, lib. i. n. 10. So Hodgson says; the reference and the fact seem both doubtful; possibly Hodgson founds upon the 6th aphorism.

<sup>3</sup> Hodgson, *op. cit.* p. 145, etc.

<sup>4</sup> *Monthly Medical Journal* (1850), p. 169.

sional success, modern physicians have almost unanimously agreed to discard this treatment, and, as we have seen, have adopted the most hazardous procedures in preference. And this for two reasons, the first of which, though really the one of least importance, is the great difficulty with which it can be carried out. In hospital practice it is almost impossible to do so efficiently, on account of the childish self-will of the patients and the facilities they have for transgressing. It is only amongst the better classes that we find men of sufficient firmness of mind fully to conform to the rigorous behests of this treatment. The only patient I have seen who fully complied with these behests was not effectually benefited, though his distressing symptoms were temporarily relieved. This difficulty about the dieting is, however, of little consequence, as most physicians are now agreed—and this is the second and most important reason why this treatment has been given up—that, fully carried out, this treatment is more apt to be injurious than remedial. From the repeated venesections the heart's irritability is increased and its pulsations are more frequent; while the long-continued rigorous diet diminishes the coagulability of the blood by causing an ultimate decrease in its fibrine. So much is this the case that Dr. Copland remarks that he has seen aneurysmal tumours which did not increase so long as the patient avoided any cause of vascular excitement and continued his ordinary diet, suddenly become greatly aggravated, increased in size, and hastened to a fatal termination, when repeated depletions were practised and vegetable or low diet adopted.<sup>1</sup> This is so consonant with physiology and with experience that I shall not multiply quotations to prove it, but content myself with recording it as the chief reason why this treatment has ceased to be employed. On the other hand we know that it is the blood-pressure that enlarges the sac and ultimately ruptures it, hence lowering of the blood-pressure must be and is the principal indication in promoting the cure of

<sup>1</sup> *Medical Dictionary* (1864), vol. i. p. 77.

aneurysm.<sup>1</sup> This important truth has been fully confirmed by the experience of modern surgeons, who have found that to cure external aneurysms by compression, it is not necessary completely to arrest the flow of blood through the tumour, but merely so to moderate the current as to make it incapable of forcibly distending the sac. Hence many physicians have adopted a modification of Valsalva's treatment, in which the importance of the recumbent position is fully insisted upon as a means of reducing the frequency of the pulse, while the diet is only so far restricted as to diminish the amount of the circulating fluid without impairing its physiological qualities. A considerable amount of success has attended this modification of Valsalva's cure, and Professor Tufnell has recorded eight cases of marked improvement, some of them indeed of apparently perfect cure.<sup>2</sup> On the other hand this treatment has not always been so successful; and even on Tufnell's own showing it is incapable of relieving the severe neuralgic pain which is so prominent a feature in many of these cases, for the relief of which various other remedies were of necessity had recourse to, with more or less success.

<sup>1</sup> Hodgson, *op. cit.* p. 164.

<sup>2</sup> Tufnell, *The Successful Treatment of Internal Aneurysm, by Consolidation of the Contents of the Sac*, second edition (Dublin, 1875). Tufnell very properly insists much on the importance of recumbence in reducing the frequency of the pulse, and of the important effect this must have upon the aneurysm. Graves considers that recumbence reduces the pulse in health from six to fifteen beats per minute, according to its rate. In debilitated and febrile conditions, the reduction is much greater.—*Dublin Hospital Reports*, vol. v. p. 561.



## LECTURE XVII

### ON THE TREATMENT OF ANEURYSM BY POTASSIUM IODIDE

ALL the methods of treating aneurysm I have hitherto described have been based upon what Tufnell has called "the consolidation of the contents of the sac." To attain this object, the most hazardous experiments have been undertaken, for, not content with the comparatively innocuous use of electrolysis, pressure, or starvation, the sac has been stuffed with iron wire, sterilised catgut, or horsehair, to provide a basis upon which the coagulum might form, but without much success. Coagulating fluids—such as the perchloride of iron—have been injected into the sac, and aneurysms have been treated by manipulation, the sac being kneaded and squeezed so as to break up any clots within it, with the view of occluding the mouth of the sac or the distal artery, or at least of providing by the broken down clots fresh nuclei for further coagulation. Some cures have been thus effected, but at great risk, and we hear nothing of the failures. It is not, indeed, ever safe to attempt to occlude the sac in this way, from the risk there is of fatal embolism attending the process. But, as I have already mentioned, there is a method of cure in which no risk is run, in which there is no attempt to consolidate the contents of the sac, but every endeavour to strengthen and consolidate its walls.

When a hollow muscle, like the bladder, is opposed to an obstacle it cannot overcome, such as an impervious stricture of the urethra, it dilates and may go on dilating till it bursts. But should the obstacle—the stricture—be one with which it

can successfully cope, it not only does not dilate, it contracts and hypertrophies. Upon this truth is based that other treatment of aneurysm I am now about to describe. In the normal condition, the coats of an artery dilate as a blood-wave passes; they contract upon it, and in thus returning to their normal state they aid the onward movement of the blood. But when the arterial coats have lost their elasticity, they yield—but with difficulty—before the blood-wave, and they contract imperfectly upon it, the circulation is hindered, and the artery itself becomes in time uniformly dilated<sup>1</sup>—it becomes a true aneurysm. In like manner when one or more of the arterial coats (usually the *media*, *vide antea*, p. 405) has been in any way locally injured, that part is unable to resist the normal blood-pressure; it dilates and forms a saccular aneurysm, which, under the continuous strain of the blood-pressure, may go on dilating till it ruptures. The only means that nature has at her command to prevent this untoward end is to fill up the sac with successive layers of clot; and art, as we have seen, has endeavoured to promote a similar cure by various procedures more or less hazardous. But if by any means we could lower the blood-pressure sufficiently, we would find that the arterial coats would behave like a hollow muscle; their natural elasticity, relieved from the distending force, would slowly contract, the tumour would gradually diminish in size, and in course of time the elastic contraction of the arterial coats would be supplemented and permanently maintained by their hypertrophy, particularly by the hypertrophy of the *adventitia*. Now this is exactly what we do by the use of potassium iodide; we lower the blood-pressure sufficiently to enable the natural elasticity of the arterial coats to contract the sac temporarily, and by and by this contraction is strengthened and made permanent by hypertrophy of these coats, and mainly of the *adventitia*. I think I shall be able to give ample proof of the truth of all these statements.

<sup>1</sup> *The Senile Heart*, p. 15, etc.

In estimating the value of potassium iodide in the treatment of aneurysm, we must ever remember that it is absolutely innocuous; risk of death or injury attends the employment of every other method of treatment that has been suggested; the use of potassium iodide alone is absolutely devoid of any danger. Further, while other measures require to be supplemented by the free use of narcotics to relieve the atrocious neuralgia which is so usual an accompaniment of aneurysm, potassium iodide relieves the pain and other symptoms so effectually and so speedily that it is sometimes difficult to get the patient to submit to any restriction whatever. So far as diet is concerned, indeed, only very moderate restriction is ever necessary—to avoid things indigestible; quantity may be safely left to the cravings of nature, while in respect of fluids the only important restriction is in regard to alcohol—the patient must be absolutely teetotal for the rest of his life. It is, as we shall presently see, a great advantage to commence the treatment with a few weeks of recumbency, but even this is not always necessary. I was acquainted with a gentleman who, in 1868, had a large and forcibly pulsating aneurysm arising from the ascending aorta and passing to the right, who was permitted to go about in moderation during the whole period of treatment. Nevertheless the improvement was so great that in 1871 there was only a feeble pulsation to be felt over the upper part of the tumour. This gentleman was under continuous treatment for a whole year, and ever after took the iodide at intervals. He was so well that for two autumns he shot regularly on the moors, and he was so active that it was difficult to restrain his movements. His unrestrainable activity cut him off at last; after a convalescence of over five years he insisted on going salmon fishing, and stood for a whole forenoon up to his waist in water on a chill October day, wielding a heavy salmon rod. The natural result occurred: he caught a bad cold; his aneurysm enlarged considerably; he had a series of fainting fits, and a serious illness

from which he ultimately recovered apparently not much the worse. Scarcely convalescent, he went off to business, not in his own comfortable carriage but in an open tramway car, caught a fresh cold, and about a week subsequently ruptured his aneurysm in a fit of coughing, and died.

I need hardly say that aortic aneurysm, even of considerable size, is not always incompatible with an active life, the pain either ceasing or at least being much modified after the chest wall has been perforated.

Some few years ago there was in Dublin a prize-fighter by profession, who had a large aneurysm projecting through his sternum; yet he suffered so little that he continued to maintain himself by his repulsive calling, only taking the precaution of putting an iron cage over the tumour when he went to battle. One of those patients whose cases are subsequently related maintained himself as a hotel porter for ten years with a large aneurysm projecting through his chest wall. Thus continued immunity from suffering, and capacity for exertion, prove nothing; they may or may not be due to treatment. But when we find a drug relieving suffering, mitigating every symptom, and restoring capacity for exertion when lost, not only occasionally but in almost every case in which it has been fully tried, all who know anything of aneurysm will agree that it must be a remedy deserving of the fullest confidence. And our confidence is increased when we find that these favourable results can be obtained altogether apart from the adjuvance of diet, rest, or of any special change in the habits of the patient; not that these things do not help, but that at times and for special reasons they may be dispensed with.

As has been so often the case in medicine, the adoption of this remedy for aneurysm has not been the result of any speculative ideas as to the nature of the disease, nor has it been based upon any known action of the drug, but has been entirely founded upon empirical observations—observations so opposed to all that was known on both of the points referred



to, that for long it was impossible to give any intelligible explanation of them. The late Dr. Graves of Dublin was the first to direct attention to the beneficial effects of large doses of iodide of potassium in painful affections of the fasciæ and nerves; and it is many years since the late Mr. Craig of Ratho informed me that a patient of his, who was taking seven grains of potassium iodide three times a day for a chronic rheumatic affection, had also obtained complete relief from the neuralgic pains arising from a large aortic aneurysm. It struck me at the time that the unexpected relief might not be accidental, and that the treatment was worthy of further trial, and I had already treated my three first cases—XLI., XLII., and XLIII.—and was about to publish them, when I became acquainted with the following history of this method of treating aneurysm:—

About 1859 a Pole presented himself to M. Nelaton with a tumour in the lower part of his neck, which had been recognised by MM. Bouillaud, Andral, and Beau as an aneurysm of the innominate artery implicating the aorta. This Pole stated that while in Warsaw his sufferings had been much relieved by the use of potassium iodide. M. Nelaton took the hint, and continued the remedy, the result being, to his great surprise, a notable amelioration of all the symptoms and an almost complete disappearance of the tumour, so that the Pole was able to return home in a satisfactory state of health. M. Bouillaud was the next to follow out this plan of treatment in four cases which he has narrated. The first was a woman with an aneurysm of the carotid artery the size of a pigeon's egg; she had fifteen grains of potassium iodide for several days, and afterwards thirty grains a day for two months. At the end of this period the tumour had so much diminished that it might be considered to have completely disappeared. The second case was an aneurysm of the brachio-cephalic trunk and aorta in a man, which, under similar treatment, underwent considerable displacement and a well-marked diminution in size. He was still under

treatment when these cases were published. The third case was a large aneurysm at the point of origin of the carotid and subclavian arteries in a man; this had considerably diminished in size after a few weeks of similar treatment. The fourth case was a carotid aneurysm in a man; it too had almost completely disappeared after a few weeks of the same treatment.<sup>1</sup>

While this problem was thus being experimentally worked out in our western hemisphere, an independent observer in the eastern hemisphere was also empirically investigating the peculiar action of potassium iodide in aneurysm, to which his attention had been casually directed, and with no less remarkable results. On commencing hospital duty in August 1860, Dr. Chuckerbutty of Calcutta<sup>2</sup> found in the wards an Irishman, aged fifty, afflicted with a harassing cough accompanied with profuse expectoration, which continued unrelieved until potassium iodide was administered in decoction of cinchona. Coincident with the relief to the cough, Dr. Chuckerbutty was astonished to find that an aneurysm of the innominate, from which this man also suffered, had gradually become solidified. This aneurysm projected above the sternal notch, and was at first the size of a walnut, with thin walls, and readily emptied; it grew steadily upwards into the neck, passing beyond the median line, till it attained the size of an orange, subsequently becoming hard and consolidated under the use of potassium iodide. Some months afterwards this man died from an attack of bronchitis, and the aneurysmal sac was found to be as large as a pear, and filled with dense coagula, which left merely a narrow channel on their outer aspect through which the right carotid and subclavian communicated with

<sup>1</sup> These cases are referred to in Dr. Roberts' paper presently to be quoted; they are given in full in the *Clinique Européenne* for July and August 1859. M. Bouillaud's cases are quoted in the *Union Médicale* of 8th March 1859, and are detailed in a clinical lecture published in the *Gazette des Hôpitaux* of the same year.

<sup>2</sup> *British Medical Journal*, 19th and 26th July 1862.

the aorta. Suspecting some causal connection between this unexpected result and the remedy employed, Dr. Chuckerbutty proceeded to treat other aneurysms in the same manner, and of these he has related three. The first of these was a man, aged forty-seven, with an immense aneurysm of the aorta, who obtained great temporary relief from the use of four grains of potassium iodide three times a day. This treatment was continued from 7th December to 12th January, when death occurred from rupture. Three hours after death the sac was found to be filled with dense, solid coagula. The second case was also a man with a large aortic aneurysm. Though only under treatment for less than three weeks, there was yet so great an amendment that the man thought himself cured, and insisted on leaving the hospital to spend Christmas with his friends. He did not return for more than three months; his symptoms were intensely aggravated, and he died in three days. The third case was a man with an aortic aneurysm, projecting as a dome-shaped tumour two inches in diameter through the sternum. Various remedies had been employed ineffectually, but under the use of potassium iodide internally, coupled with the application of tincture of iodine externally over the tumour, the patient steadily improved. The thoracic pain ceased; the tumour diminished; the hæmoptysis ceased, and the patient ate and slept like any other man. He was still under treatment when the paper was sent home for publication. Dr. Chuckerbutty points out that the important fact in the history of these cases was the consolidation of the contents of the sac, and contends that this depends upon some hitherto unsuspected action of potassium iodide.<sup>1</sup> This opinion seems also to have been shared by Dr. Roberts of Manchester, to whose interesting paper I shall now refer.

About the beginning of 1862, Dr. Roberts was consulted by Mr. T. Windsor in regard to a case of aneurysm, and his

<sup>1</sup> This very natural mistake as to the action of potassium iodide has done much to retard the usefulness of this important method of treating aneurysm.

attention for the first time directed to the important remedial action of potassium iodide in that disease. The patient was a lady, aged twenty-nine, who had an aneurysm of the aorta implicating the origin of the innominate. There was excessive pulsation at the root of the neck on the right side, repeated slight attacks of hæmoptysis, occasional loss of voice, dysphagia, constant troublesome cough with scanty expectoration, and recurrent paroxysms of pain and dyspnœa so severe as to compel her to get up and walk about. Under a regulated diet and strict confinement to the recumbent posture her condition became steadily aggravated. She became so weak and emaciated as to be unable to leave her room; the right clavicle projected half an inch beyond its natural level, and the pulsation was much increased. In April 1862 she was put upon potassium iodide with the view of relieving the severity of the pain, and this object having been attained the remedy was discontinued, having only been employed one week. At last, in July 1862, when she was so much worse that death seemed not far off, Mr. Windsor put her upon five grains of potassium iodide three times a day; this was subsequently increased to ten grains, and again reduced to five on account of profuse salivation. The five-grain dose agreed, and the patient continued taking it up to the time of the publication of the case.<sup>1</sup> The result of the treatment was a rapid subsidence of all the general symptoms, complete cessation of cough, pain, dysphagia, and hæmoptysis, while the patient gained flesh and strength and in a few months was able to walk six miles; the clavicle also subsided to its normal position. Dr. Roberts<sup>2</sup> subsequently treated in the Manchester Royal Infirmary a man, aged thirty-nine, in whom the first bone of the sternum and its vicinity were the seat of heaving pulsation, while in the second left intercostal space there was a conical, soft, pulsating elevation, projecting about a quarter of an inch, and with a base the size of a shilling. Over the

<sup>1</sup> *British Medical Journal* for 24th January 1863.

<sup>2</sup> *Loc. cit.*



bulging part there was an area of dulness measuring transversely four inches and a half, and vertically three inches and a half. There was pain in the left side of the head and shoulder, dyspnœa, dysphagia, and some cough. This patient was put upon a restricted allowance of fluids, and had five grains of potassium iodide given him three times a day. In three days the dose was raised to seven and a half grains three times a day. In six days the patient declared himself to be much better, his pain gone, and the cough and difficulty of breathing less troublesome. The dose of the iodide was then raised to ten grains three times a day, and after seventeen days' treatment the pain, the dysphagia, and dyspnœa were quite gone; the pulsating tumour had entirely subsided; the patient was allowed to get up, and his restrictions were relaxed. The dose was now increased to fifteen grains, and after twenty-five days' treatment to twenty grains thrice a day. After this dose had been continued for about five days it was reduced to five grains on account of the supervention of diarrhœa. The bulging was decidedly less; the area of dulness was reduced to three inches and a half transversely, by two inches and a half vertically. The projecting tumour had quite disappeared, and the general pulsation was almost gone; but the enlarged superficial veins, and the stridulous voice, still remained to show that the cure was not complete. In this case decided emaciation was the accompaniment, if not the result of the treatment. Dr. Roberts<sup>1</sup> also relates a case which occurred under the care of his colleague, Dr. Wilkinson, of a large thoracic aneurysm in a man, projecting in the neck as a tumour the size of a child's head, extending from the left clavicle to the angle of the jaw, and apparently about to burst. Fifteen grains of potassium iodide were administered three times a day; the pain was lessened, and the growth seemed stayed, but the patient died in seventeen days from gangrene of the lung, the result of pressure upon the left

<sup>1</sup> *British Medical Journal*, 24th January 1863.

pneumogastric nerve. The sac was found lined with coagula, and a large, firm blanched clot, attached by a broad pedicle to its upper part, floated free within it.

During the last thirty years, I have employed the potassium iodide in the treatment of aneurysm with unvarying success so far as relief to symptoms is concerned, and with such favourable results in retarding the progress of the case, and even, in some instances, promoting an apparent cure, as certainly stamp this treatment as the most efficient hitherto propounded for the relief of this intractable complaint, while it has the further advantage of being in itself wholly innocuous. On former occasions I have narrated in full a number (eleven in all) of cases treated by this method, but it is quite needless to enter into so much superfluous detail; I shall therefore content myself with giving such an abstract of these cases as will suffice to show their character, and to enable us to trace the manner in which the true action of the potassium iodide was at first surmised and finally proved, so far as such remedial actions can be proved.

CASE XLI. Peter Rice, a mason, aged thirty-nine, admitted into Ward III. on 29th April 1867.<sup>1</sup> Patient has never had rheumatic fever, but for five years past has been subject to pains in his hip, leg, and back, which are severe and most apt to recur during the changeable weather of spring. He has been in the habit of drinking freely. About twelve months before admission he fancied he was overwrought, and when he got home he found he could not take a deep inspiration with the usual freedom. He also felt a sharp pain at a point about two inches to the right of the left nipple and a little above it; this pain has continued ever since, and has latterly increased considerably. This pain is stationary at the part described, but when more than usually

<sup>1</sup> This case was at first under the care of Dr. Warburton Begbie, acting for Professor Laycock, and was transferred to my care when the clinical wards were closed at the end of the summer session in 1867. The case is partly taken from the clinical records of Ward III.

severe it spreads upwards to the arm-pit and shoulder, and down the left arm to the wrist; occasionally it extends to the *scrobiculus cordis*, and sometimes strikes sharply through to a corresponding point at the back. The pain is specially apt to be severe at night, preventing sleep, and is very annoying when it affects his back, as he cannot lie in any other position than the supine. He has occasional fits of dyspnœa, and it is always painful to take a full inspiration. He also has some difficulty of swallowing. He is much troubled with palpitation and a distressing feeling of pulsation within the chest, and these sensations annoy him most when the pain is severe. At these times he gets some relief by relaxing the respiratory muscles by leaning forwards and resting against a wall or other support, with his hands and arms extended. About a week ago his symptoms had so much increased that he was compelled to cease working entirely. His appetite is good; his bowels generally confined. On percussion, the heart seems of normal size; its pulsations are distinct, and in their ordinary situation; the first sound is normal, the second accentuated. The right radial pulse is fuller than the left. About three years ago the patient was troubled with *muscæ volitantes*, lasting off and on for about two years, and appearing only for a few seconds each time. He still at times cannot see things at a distance so well as he thinks he ought, and the letters seem to swim before him when he reads. His left pupil is slightly dilated. Some time ago he had *tinnitus aurium*. His left cheek is often flushed, and at times he feels it warmer than the other. His lung sounds are normal, but he has an imperfect, hard, clinking cough, without expectoration. He also frequently perspires without any apparent cause. There is a slight bulging of the walls of the chest between the second and third ribs, at the left edge of the sternum, extending into the *manubrium sterni*, and gradually declining all round, within an area equal to that of the mouth of a tumbler. There is dulness on percussion over this bulging, and a distinct feeling

of liquid pulsation. Within this area the heart sounds are extremely distinct, but there is no murmur.

Twenty grains of potassium iodide were given three times a day, and a belladonna and opium plaster was applied over the tumour. About a month subsequently a similar dose of the bromide of potass was substituted for the iodide, and at first he thought himself benefited by the change, but in about a week the iodide was again had recourse to with the addition of one-twelfth of a grain of iodine, but in another month (7th July) the iodide was stopped on account of coryza. It is not known whether recourse was again had to the iodide while the patient was in Ward III.; it is, however, believed that it was. The last entry is on 18th July—"Thinks himself easier to-day." On 1st August the patient was transferred to Ward VII. and placed under my care. Rice was at once given thirty grains of potassium iodide three times a day, and these doses he continued to take without intermission up to May 1868, with continually increasing benefit, and without the production at any time of the slightest unpleasant symptom. For several months he wore a belladonna plaster over the tumour, but at last it caused so much eczema that it had to be given up. On coming under my charge he was strictly confined to bed, and for long he lay entirely on his back, that being the only position in which he always found comparative, and latterly perfect ease. He was not allowed out of bed till 2nd April, about three weeks before his discharge. At first he was fed on fish, meat being subsequently given when he tired of fish, but he was warned to be strictly moderate both in eating and drinking; water, tea, and milk, being the only fluids supplied. Rice's progress was extremely slow at first, but it was steady. In a clinical lecture on his case on 19th November 1867, it is stated, "There is no longer any tumour visible, and it is only on careful examination that you will discover any pulsation. He has so far recovered that he is inclined to be rash, to move about quickly in bed, and even to turn upon his side;



but upon this the pulsation instantly returns—a sufficient warning to him that he is not yet cured.” It was not till the beginning of March 1868 that he was able to move about freely without discomfort, and without return of pain or pulsation. On 1st April 1868 I showed him to the Edinburgh Medico-Chirurgical Society, just previous to his discharge from hospital, and its members had an opportunity of observing the complete subsidence of the tumour described, pulsation being only obscurely felt at the spot. They also heard the man’s own statement that his pain, dyspnœa, and dysphagia were gone, and they were able from his healthy and energetic appearance to form some idea of the importance and value of the treatment to which he had been subjected.

A year afterwards (1869) I reported to the Medico-Chirurgical Society as follows:—“Peter Rice, labouring under aneurysm of the aorta, has been repeatedly under observation during the past twelve months. When first discharged, he acted as night watchman for a month or two; he was then appointed to an institution for the care of orphan children; he walked about with them and took them to and from school; latterly he set up a small shop as the easiest way of making his living. He continued to take the potassium iodide, but not regularly, and his aneurysm remained as it was when he was discharged; the same dull thud audible in the second left intercostal space, but no pain nor any uneasiness.”<sup>1</sup>

Rice continued to present himself occasionally, till, after a longer interval than usual, I found him in his working clothes laying pavement on the street. He continued to employ himself at his old trade of a mason for two years, and his ability to pursue this laborious occupation for so long and without suffering is sufficient proof of the reality of his amendment. It is something surely akin to a cure to be able to restore to so much usefulness one who so shortly before had been crippled by the agonies of so terrible and,

<sup>1</sup> *Edinburgh Medical Journal* (July 1869), p. 47.

till then, so hopeless a disease as aortic aneurysm. Rice died suddenly in the summer of 1872, while working in the Botanic Garden; he caught a bad cold some weeks previously, and thinking and feeling himself otherwise quite well, he took no care but continued to work as a labourer up to the moment of his death. The immediate cause of death was hæmorrhage into the pericardium from a very minute rent at a comparatively thin part of the aneurysm, just where it sprang from the aorta. The aneurysm itself was the size of a small cocoa-nut, its walls everywhere dense and firm, save just at the part where the rupture took place; it contained only *post-mortem* clots. At the very outset of our experience, we had thus a most remarkable case, in which the sufferer, without risk to life, and with speedy relief to suffering, was so restored as to be able to maintain himself in comfort for four years. The condition of the aneurysm was also instructive, it was contracted, its walls were dense and firm, and it contained only *post-mortem* clots.<sup>1</sup> It was in every way a most instructive and satisfactory case; but we had much yet to learn.

My next case, though more obscure in its diagnosis, was even more immediately satisfactory in its results.

CASE XLII. John Kerr, a seaman, aged twenty-six, admitted into Ward VII. on 22nd October 1867. He said he had been ill for eighteen months, dating his illness from the time of an arctic voyage, when he had been exposed to much privation and had to make strenuous exertion in hauling boats over ice and snow while in a feeble state of health. In America he had been supposed to suffer from an aneurysm of the abdominal aorta, and he presented himself at the surgical wards of the Edinburgh Royal Infirmary to be relieved of this, thence he was transferred to my care. Kerr complained of intense pain in the *scrobiculus cordis* extending through to the back and passing round both sides. On ex-

<sup>1</sup> The preparation is in the Museum of the Royal College of Surgeons, Edinburgh.

amination a tumour could be obscurely felt a little below the sternum, and just under the edge of the right false ribs. On turning the patient over toward the left the tumour was more readily felt; it dropped, as it were, into the hand. Over the tumour referred to, or at least as near as could be got, a loud murmur was to be heard through the stethoscope. Notwithstanding the obscurity of the diagnosis, it was thought desirable to give this patient potassium iodide, as likely to be useful whether the tumour was solid or truly aneurysmal. Accordingly he got thirty grains of potassium iodide twice a day; he was put upon fish diet, a restricted amount of fluids, and confined to bed. The result was remarkable; he got almost immediate relief from the agonising pain, the uneasy pulsation felt by the man himself was at once considerably lessened, and the force and volume of the radial pulsations appeared to be also greatly diminished. He improved steadily; in a few months tumour and murmur had quite disappeared, and on 22nd January he was discharged at his own request, and engaged himself for a short voyage to test his reacquired health before going to sea for a longer term. The obscurity of this case is patent to all, yet the signs and symptoms pointed strongly to aneurysm, and the success of the treatment, coupled with the rapid manner in which relief was obtained, seemed to confirm the diagnosis.

A year afterwards I reported as follows:—"John Kerr left for a trial voyage, but never returned. In January 1869, a year after his discharge, the ward nurse got a letter from him dated Australia and saying he was now so well that he was off to the gold diggings."<sup>1</sup> I have heard nothing more of this patient.

The diagnosis in the next case was more unequivocal, and as the history includes the *post-mortem* appearances, we know exactly what ailed him and to what extent it was remedied.

CASE XLIII. James Wilson, a mason from Newcastle, aged forty-four, admitted into Ward VII. on 31st August

<sup>1</sup> *Edinburgh Medical Journal* (July 1869), p. 48.

1867. About nine months before admission this patient began to have occasional attacks of lightness in the head with a flashing of light before his eyes. These attacks came on while he was at work, and obliged him to sit down for a little to recover himself. At first the seizures came only once or twice a day, but they soon became more frequent, and he always felt much weaker after them. During these attacks he perspired profusely. About the same time the patient began to suffer from "beatings" in his abdomen, in the left side of the chest, and on the right side of his neck. A swelling also appeared at the right side of his neck which gave him great uneasiness, and produced a feeling of choking. Getting no relief from those whom he consulted he came to Edinburgh. On admission to the Infirmary it was found that he had no pulse in the left radial artery, but for this there was no apparent cause, the "beatings" on the left side being evidently only cardiac palpitation; on the other hand, those in the abdomen, and on the right side of the neck, had each an abnormal and evident cause. In the lower part of the epigastric region, towards the left side, and lying close above the aorta, the course of which could be distinctly traced, a tumour the size of a small orange could be distinctly felt pulsating—expanding in all directions—and not merely moved by the artery beneath it. Over this tumour a loud systolic murmur was heard. On the right side of the neck there was also a pulsating tumour extending up the neck from the sterno-clavicular articulation towards the mesian line. This tumour was somewhat larger and longer than that in the abdomen; it resembled in shape a large kidney potato, and upon any excitement, especially when the man was up and walking about, it increased considerably in size. No distinct murmur could be heard over it, nevertheless it was evidently an aneurysm implicating the innominate, subclavian, and carotid arteries. Wilson was at once given thirty grains of potassium iodide twice a day, and a diet and regimen similar to that already described were prescribed for him,



but as his symptoms were not so urgent strict recumbence was not insisted upon, and he was allowed to go to chapel every night. The iodide was not so well borne by this patient as by the two already commented on; every now and then pain in the stomach, or severe headache gave warning that it was time to omit it. These symptoms always abated after leaving off the medicine for a day or two. His appetite always remained good, but his bowels required to be regulated by medicine. No immediate effects were observed from the iodide, but after some months the abdominal aneurysm was found to be quite firm and apparently solid; the murmur had also disappeared, and could only be reproduced by pressing somewhat strongly with the stethoscope. After a time the aneurysm in the neck also ceased to swell out when he walked about; it gradually became firmer and ceased to give him any inconvenience. His general health was much improved; he no longer had any disturbing pulsations, and though he occasionally suffered from lightness in the head, and from dazzling flashes of light, his condition was much better than it had been, and, considering his inveterate aneurysmal diathesis, he had probably reaped as much benefit from the treatment as was possible.

A year subsequently I reported of him as follows:—“James Wilson, labouring under an aneurysm of the innominate, implicating the subclavian and carotid arteries, also an aneurysm of the abdominal aorta, and a general diseased state of the vessels. This man worked for many months comfortably and without inconvenience at his trade of mason, avoiding, as desired, any heavy lifts; he has now, however, got an easier berth in connection with the Caledonian Railway. His abdominal aneurysm is still to be felt as a hard, firm knot, much diminished in size from what it was. His innominate aneurysm never troubles him, and gives rise to no symptoms, but it is not consolidated, neither is it any longer an aneurysm. To all intents it is nothing now but an elastic artery, fusiformly dilated no doubt, but

no longer bulging as a globular pulsating tumour across the trachea.”<sup>1</sup>

This poor man presented himself every now and again for inspection, and occasionally came into hospital for a week or two for relief of catarrhal symptoms. On 27th June 1870 he re-entered hospital because of a recurrence of the symptoms of abdominal aneurysm. The original abdominal aneurysm was to be felt as a small hard nodule resting upon the aorta just beneath a large, soft, pulsating swelling passing up beneath the ribs on the left side. Over this tumour a systolic murmur was to be heard; the subjective symptoms were confined to flatulence and occasional pain of a dyspeptic character in the stomach. He was treated as formerly with potassium iodide and with a similar result. By the beginning of December 1870 he was about to be discharged when he showed symptoms of typhus, caught through unauthorised contact with fever convalescents in the infirmary chapel. He was removed to the fever wards, passed through the regular course of typhus, and after disinfection was received back into a side-room seven weeks subsequent to his transference. On 24th January 1871 his abdominal tumour measured six inches, from the lower edge of the hard nodule of the first tumour to the point where it disappeared beneath the ribs. The tumour lay along the aorta; it was three inches in breadth, and could be readily grasped through the thin abdominal parietes; it was firm and devoid of lateral pulsation; and, unless firmly pressed upon by the stethoscope, no murmur was audible, merely a dull thud. At its upper extremity, however, just where it dipped beneath the ribs, a slight systolic murmur was still to be heard, and this part of the tumour seemed to have grown somewhat larger during his illness. Wilson made a good recovery, was discharged, and continued to maintain himself by manual labour till September 1873, when he was readmitted to the Infirmary for a recurrence of symptoms of aneurysm, and he died there on

<sup>1</sup> *Edinburgh Medical Journal* (July 1869), p. 48.

14th October, over six years after his first admission. The large, recent abdominal aneurysm occupied the whole of the left hypochondriac region; at first its pulsations were quite fluid, but under treatment the walls of the sac became firmer and the pulsations less distinct, but it never diminished in size; indeed for this there was scarcely time before the patient died exhausted. After death the whole of the aortic arterial system was found to be greatly atheromatous; the innominate, subclavian, and right carotid arteries had thicker walls than usual, and seemed altogether larger. The left subclavian was plugged by a firm fibrinous clot just where it passed out of the chest, the coats of the artery being contracted round this plug. The descending aorta gradually dilated till, after passing through the diaphragm, it developed into an aneurysm the size of a large cocoa-nut, and below this there lay the hard, firm nodule of the original aneurysm; this contained only *post-mortem* clots, but its walls were made almost as dense and hard as bone by atheromatous deposit.

CASE XLIV. Peter Reid, hotel porter, aged forty-six, admitted into Ward VII. 18th June 1868. This man was drunk on admission, having avowedly taken spirits to nerve himself for his apparently rapidly approaching dissolution. He had intense dyspnoea, amounting to complete orthopnoea, violent, harassing, but dry cough, and felt and looked as if on the point of suffocation, while a large, soft, projecting and pulsating tumour seemed to threaten death in another form. He was ordered at once fifteen minims of chlorodyne to be repeated every half-hour till the cough was quieted, and also twenty grains of potassium iodide three times a day. He was confined to bed, in which he was supported in a semi-erect posture, and his food and drink were somewhat restricted. Within twenty-four hours this man expressed himself as much relieved, and in a few more hours he breathed more freely. Reid was an old soldier, and about five and twenty years previously he had been cupped in a military hospital for palpitations which continued more or less after his discharge. About

ten years ago, in the end of February 1858, he was admitted into this hospital on account of excruciating pain in the right side and down the right arm, which had troubled him for some months previously. There was also at that time a slight swelling, accompanied by pulsation, to the right of the sternum. The pain complained of was most excruciating in winter, when he caught cold, or made any unusual exertion. In about eighteen months the tumour protruded externally, and then the pain ceased to be so agonising. During the past eighteen months the tumour has grown more rapidly than it has done for years; this he attributes to the extreme violence of his cough.

To the right of the sternum there was a large pulsating tumour, extending from the third to the seventh rib, and projecting fully half an inch beyond the level of the thoracic wall. Part of this tumour was solid, but part, rather beneath the middle, was soft, painful to touch, projecting conically, and pulsating fluidly. Dulness extended for fully five inches round the centre of the tumour, passing below into the liver dulness. The pulse in the right arm was smaller than that in the left. The heart's apex beat between the sixth and seventh ribs and one inch to the left of the nipple, but there were no indications of hypertrophy, and the cardiac dulness was normal. There was cardiac displacement but no hypertrophy, and there were no abnormal murmurs to be heard either over heart or tumour. The right pupil was somewhat dilated. There was considerable pain in the right side and down the right arm, but this was much less than formerly. After the patient had somewhat recovered from the frightful condition he was in when admitted, a belladonna plaster was applied over the tumour, and the chlorodyne was gradually stopped as the cough lessened. It had almost entirely ceased by 12th August, at which date the patient looked, and expressed himself as feeling much relieved. The pulsation in the tumour was much less forcible than formerly, and the tumour itself was flatter and felt more solid. On 31st



August an icebag was substituted for the belladonna plaster for a few days, but had to be given up, as, though it felt comfortable enough and relieved the distressing pulsation, it increased the cough very much. At this date he expressed himself as feeling very comfortable, and able to breathe and swallow easily, both of these acts having been hitherto attended with much uneasiness, the tumour was also gradually decreasing in size.

Reid was kept in hospital till 20th March 1869, when he was discharged to go to his home in London. Thus this poor dying creature was, after nine months' treatment, able to be discharged in a comparatively active condition, and both looking and feeling well. A cast taken from the projecting tumour about a month after admission, and one taken on the morning of his discharge, show the marked diminution in its bulk. In a letter from my resident, the late Mr. Frank H. Hodges, he says, "I called on Peter Reid on 28th April, and found him in a very satisfactory condition; the tumour has considerably diminished in size (since his discharge); he is quite free from cough, and goes out for a constitutional daily."

The next case is evidently not one of saccular, but rather of true aneurysm (dilated aorta) with diseased arterial coats, and gives very evident proof of what can be done for the relief even of these cases by the treatment recommended.

CASE XLV. Thomas Moody, aged thirty-nine, a slater from Crossgates, Fife, admitted to Ward VII. on 11th July 1868. He stated that for sixteen months he had complained of severe pain across the upper part of the sternum, and a feeling of breathlessness. On percussion dulness was marked across the whole of the upper part of the sternum. The cardiac dulness was normal, or nearly so; the apex beat in the normal position between the fifth and sixth ribs. The first sound was normal, the second wanting and replaced by a double murmur; the second or diastolic portion of this was loudest at the aortic cartilage (the second right). This

double murmur was propagated upwards into the arteries, and its systolic portion was louder and rougher over the left carotid artery than anywhere else. The finger pressed deep into the tracheal fossa came in contact with a pulsating body. The other organs and systems were natural.

Moody got thirty grains of potassium iodide three times a day; he was confined to bed, and had his food and drink somewhat restricted.

The patient very speedily expressed himself as greatly relieved. In a fortnight the rasping murmur over the left carotid was much softened; the pulsation in the tracheal fossa was still perceptible, but not so distinctly as formerly. He was discharged at his own request on 3rd August 1868. In this case there was no history either of rheumatism or syphilis.

The case now to be related, on the other hand, is not only a well-marked but also quite a remarkable case of aneurysm of the aorta, one of itself quite sufficient to attract attention to the treatment propounded, and which, as one of a series, affords indeed a very marked illustration of the benefits to be derived from it.

CASE XLVI.<sup>1</sup> Andrew Jamieson, a carter, aged forty, admitted into Ward II. under Professor Bennett's care on 31st January 1868. About eighteen months before admission this patient had an attack of pleurisy on his left side, but with this exception he had been always healthy. About twelve months ago, without known cause—as he is not specially aware of having strained himself, though constantly in the habit of lifting heavy weights into his cart—he began to feel a severe and constant aching in the left side of his chest, over a spot about the size of a crown piece about two inches above the nipple. This pain was much increased on exertion or on stooping. To relieve this pain he unavailingly applied several mustard poultices, and took a quantity of

<sup>1</sup> The history of this case is partly condensed from the Clinical Records of Ward II.

cod-liver oil. Notwithstanding his sufferings he continued at work till July 1867, but was then compelled to give in. The pain was now more severe than ever, his breathing short and wheezing; he had a troublesome cough, worse upon exertion; he had a choking feeling when he stooped, and a difficulty in swallowing any solid food, the bolus seeming to stick just opposite the upper margin of the sternum. His voice was now also weak and somewhat hoarse—*vox anserina*—and he felt a disagreeable pulsation in his chest. For these symptoms he sought relief in Glasgow Infirmary, and finally came to Edinburgh. Up to the period of his admission the pain had spread very much across the upper part of his chest, but he does not think that the other symptoms had increased; he has never had either headache or hæmoptysis. The left radial pulse is almost imperceptible. The right radial pulse is 84, of moderate strength, and slightly jerking. Cardiac dulness not noted. The heart's apex beats three inches below and a little external to the nipple line. Only the first sound is heard at the apex; at the base both sounds are audible and are normal. Over the upper part of the left chest anteriorly there is a distinct bulging, most marked over the second rib and second intercostal space. Over a spot about the size of a crown piece there is a distinct impulse communicated to the stethoscope. Over the left chest there is dulness on percussion from the clavicle to within two inches of the nipple line; this dulness extends across to the opposite sterno-clavicular articulation (*vide* Fig. 34). Over this dull area a double blowing murmur is to be heard, "the second sound, however, being very feeble, and heard most distinctly over the sternum, opposite the articulation of the second rib, also heard over the great vessels at the root of the neck."<sup>1</sup> At the right pulmonary apex the inspiration is harsh and the expiration prolonged. Over the left apex

<sup>1</sup> I quote the *ipsissima verba* of the report. The words "second sound" may have referred to the diastolic murmur, but as the points alluded to are off the tumour, the statement may be quite correct.

anteriorly the double murmur referred to completely obscures the sounds of respiration; everywhere else they are normal. The patient has a frequent and loud cough, of a clanging, metallic character, without expectoration. He complains of severe, dull aching pain on the left side of the chest, from below the clavicle to the level of the nipple; this pain occasionally extends to the right side. He gets only occasional short snatches of sleep, partly owing to the pain referred to, and partly to the frequent recurrence of a choking sensation referred to the throat. His tongue is clean, appetite bad, but bowels regular. Urine, specific

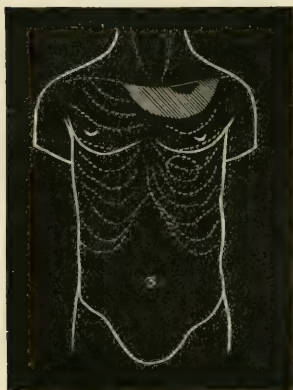


FIG. 34.

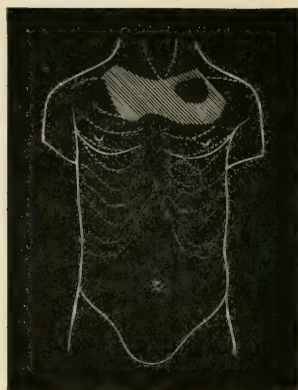


FIG. 35.

gravity 1·030, with deposit of urates, but otherwise normal. There is a slight puffiness of the integuments over the upper part of the sternum, and the veins over the dull area are visibly enlarged.

Up to 17th February his treatment was limited to the use of narcotics for the relief of the cough; at that date he was put upon twenty grains of potassium iodide three times a day; this he continued to take, though somewhat irregularly, and the report on 9th June is—"Since last report he has continued much the same. He sometimes complains of pain in the chest, still has cough with profuse purulent expectoration, and loud sonorous rhonchi both anteriorly and posteriorly.



He continues to take the sedative draught and iodide mixture. On percussion the dulness is found to have extended more towards the right side, now reaching to about the middle of the clavicle (*vide* Fig. 35)."<sup>1</sup> On 23rd June it is stated that the patient complained of great pain in the chest, which seemed to be increased, and for which he frequently had poultices applied. He also complained of great weakness, for which he got two ounces of port wine daily. His breathing was, however, easier, and the cough and expectoration greatly diminished. The pulsations over the now greatly enlarged dull area are, however, much increased, and the double blowing murmur is more distinct than formerly. Posteriorly the blowing murmur can also be distinctly heard about the sixth dorsal vertebra. 20th June.—Complains of pain in the left side being increased; poultices ordered. 27th June.—Still complains of great pain in his left side; for this he got a hypodermic injection of about one-third of a grain of morphia. He continued to improve slightly. On 16th July it is stated that the patient feels very well, has little cough or expectoration; he takes his food well and has little pain. On 23rd July the patient is said to be continuing better, his breathing easier, cough and expectoration almost gone, and for the last two or three days he has been walking about the ward, though not yet able for much exertion. He continues the use of the iodide. At this time, Ward II. being closed for the autumn, he was put under my care in Ward VII. Shortly after this he walked the whole length of the ward to the water-closet, which is somewhat draughty; this was followed by a return of his cough, copious purulent expectoration, and violent pain in the left shoulder and over the large pulsating tumour, on the left of which a small projection of about an inch square was thrown out; over this there was a red blush. Jamieson was strictly confined to bed, a belladonna plaster

<sup>1</sup> Figs. 34 and 35 have been accurately copied from drawings by Dr. Bennett's clerk. The dark spot to the left indicates the place where the red projection subsequently to be mentioned was thrown out.

was applied over the tumour, thirty grains of potassium iodide given three times a day, and an opiate mixture for his cough. In a few days his symptoms were much relieved. A further exertion, however, coupled with exposure to the damp air of a newly-washed ward, brought on a violent exacerbation of the cough with a great increase in the size of the tumour, which was wholly covered by a red blush. There was complete loss of pulse in the left arm, accompanied by coldness and excruciating pain in the limb; the expectoration was copious and purulent, and occasionally both streaked and stained with blood. His left arm was ordered to be swathed in flannel; the tumour to be covered with a belladonna plaster; to take his iodide mixture four times a day; to continue his cough mixture, and to have fifteen minims of chlorodyne *pro re nata* as often as required. The order being to keep the cough quiet at all hazards, he took for many days two drachms of chlorodyne in the day besides an opiated cough mixture. (For this see Christison's *Dispensatory*, p. 839.) He bore this treatment well; his tongue kept clean and his appetite fair. In eight days the expectoration began to lessen, and the cough and pain to cease. By 31st August the pain and cough were almost gone, and the expectoration reduced to a trifling quantity of mucus; the left arm was again warm and feeling normal, and the pulse had returned to the wrist, though it was still feeble. The tumour was diminished in size, and apparently not pulsating so strongly. He was lying quietly in bed reading, and expressed himself as feeling a new man. For the next nine months Jamieson was kept on his back, mostly on a water-bed; his cough was kept quiet by a free use of opiates, and he had two drachms of potassium iodide daily, except for a couple of days on two occasions when it had to be omitted on account of gastric irritation, as evinced by pain and vomiting. At last, towards the end of May, the tumour seemed sufficiently consolidated, and all the symptoms so moderated as to warrant the patient's being permitted to get out of bed. At this time his pulse was 100,

much fuller in the right radial than in the left; his skin moist; his cough so slight as to be inappreciable, and rarely heard during visit; his expectoration merely a trifling catarrhal mucus; his voice still thin, and somewhat feeble, but distinct and very different from what it was; respiration everywhere natural, and the percussion note posteriorly normal. The heart's apex was felt beating between the fifth and sixth ribs a little outside the nipple line, the heart being probably elongated and certainly displaced, the lung entirely covering it. In the parasternal line just outside the left edge of the sternum dulness extended from the liver dulness right up to the lower border of the first rib. In the nipple line dulness extended transversely to a distance of two inches from the left edge of the sternum. Just at the inner edge of the left clavicle there is a small patch of clear sound; with this exception, dulness extended along both clavicles, from about one inch and a half to the right of the right edge of the sternum to about four inches to the left of its left edge. The dulness on the right side is bounded by a semicircular line joining the sternum at the upper border of the second rib. On the left side the dulness is also bounded by a semicircular line the centre of which is about the middle of the left clavicle, and in this position it does not descend lower than the middle of the third rib. The sternal ends of both clavicles were dislocated; the right to the extent of half an inch, the left to about a quarter of an inch. On the upper part of the sternum there is considerable puffiness, and over it and over the dull portion to its left there are many large and tortuous veins. A finger pushed behind the sternum, in the tracheal fossa, feels only a solid mass. Over the apex beat the first sound of the heart is heard somewhat obscured by the rough systolic portion of a double murmur, heard loudest at the middle of the sternum just at the junction of the fourth rib. This murmur is propagated upwards loudly and rough into both carotids; less distinctly into the right subclavian, while in the region of the left subclavian, over the

dull part mapped out, we have a solid tumour pulsating, but not forcibly, and only with a movement of elevation and none of expansion, over which no murmur whatever is to be heard, merely a dull thud. The patient has now no difficulty in breathing or swallowing; he walks up and down stairs and about the airing ground; he says he has now no cough. Shortly after this Jamieson was sent downstairs to Dr. Bennett, who exhibited him to his class, and, as I am told, expressed himself satisfied with the reality of the improvement which had taken place.

Jamieson was discharged shortly afterwards. He went to the Workhouse, where he remained till 1st November 1869. At that date he was readmitted to Ward VII. for some catarrhal symptoms, his aneurysm being *in statu quo*. A thick oedematous collar full of tortuous veins had developed at the lower part of his neck and over the upper part of the sternum. He was discharged in April 1870, and went to Glasgow, where he remained much the same when last heard of.

This very interesting case affords an instructive example of the good that may be done by careful treatment even in the most unfavourable cases of aneurysm, and it speaks well for the treatment under consideration that it was capable of relieving symptoms so severe as those of Jamieson, and of prolonging life in circumstances apparently so hopeless.

I might easily multiply histories of aneurysms treated by potassium iodide,<sup>1</sup> but a sufficient number has been given to illustrate the nature of the relief obtained. I have treated after this fashion a very considerable number of cases of aneurysm; most of those not related were aneurysms of the aorta and chiefly of its thoracic portion; a few indeed spat blood, and in them the aneurysm seemed to be weeping; in all there was a similar result—speedy relief to pain and suffering, and in a few there has been so complete a subsi-

<sup>1</sup> In former editions of this work five other cases have been given in full, but it seems quite unnecessary to repeat them.



dence of the tumour and relief to all symptoms, as to amount to an apparently perfect cure. These results are extremely encouraging, and when we reflect upon the entire absence of any risk to the patient, the certainty of relief to his sufferings, and prolongation of his life, I think I am warranted in saying that no treatment of internal aneurysm hitherto devised holds out anything like an equal prospect of relief—if we cannot call it cure—as that by potassium iodide.

Some have supposed that the benefit derived from potassium iodide in the treatment of aneurysm is due to its anti-syphilitic virtues. I am not prepared to accept a causal connection between syphilis and aneurysm as so invariable as to make this at all likely, and even if this were true I cannot conceive what possible connection there can be between the *modus operandi* of the drug as I understand it, and shall presently expound it, and any anti-syphilitic virtues it may possess.

Dr. Chuckerbutty supposed the iodide to increase the coagulability of the blood, and Dr. Roberts seemed to consider this a plausible theory. This, however, is quite contrary to all that is known as to the action of potassium iodide, which is believed to induce quite an opposite condition of blood—a greater fluidity. Nor is this idea consistent with the results of *post-mortem* examinations, because, though we occasionally find the sac full of coagula, we learn from such cases as Rice and Wilson that clot formation in the sac is only a concomitant and not an essential phenomenon. Nay, more, many *ante-mortem* experiences convey the same lesson. How often have we found men with large and thin-walled aneurysms, which seemed to grow not only smaller but also firmer and harder under treatment, as if lined by dense coagula, grow weary of the restraint of hospital life, discharge themselves, and after a period of loose living return with all their former symptoms and with the aneurysmal pulsation just as fluid as ever. This transformation was often so rapid as to leave no doubt that the previous firmness and feeling of density of the sac had not been due to solid coagula within

it.<sup>1</sup> That coagulation of the blood forms no part of the action of this drug was also well shown in the case of a sea-captain<sup>2</sup> who was under treatment, in Ward V, for a very tense innominate aneurysm projecting into the neck. He had only been a few days under treatment when the aneurysm suddenly ruptured, the blood apparently escaping just beneath the *adventitia*, forming a dissecting aneurysm. A large, livid, pulsating tumour, the size of a child's head, lay at the root of the neck, and a boggy pulsation could also be felt between the two first intercostal spaces. Fearing external rupture I sent for my colleague, Dr. John Duncan, to endeavour to prevent this catastrophe by galvano-puncture. By the time Dr. Duncan arrived all pulsation in the tumour had ceased; no interference was attempted, and nothing was done beyond continuing full doses of the iodide. The whole of the effused blood, which remained fluid up to the last teaspoonful for a period of eight weeks, was slowly absorbed. When this was effected all trace of the aneurysm was found to have disappeared, and the man was discharged. Some weeks afterwards he returned for inspection in apparent health, and without any recognisable trace of his aneurysm or any return of his symptoms. He subsequently took command of a small vessel belonging to himself, and died suddenly in the West Indies a year or two afterwards, possibly from the rupture of another aneurysm. The case of Wilson, however, whose aneurysm, as I have mentioned (p. 452), could be easily grasped through the abdominal parietes, and was felt to grow firmer and harder as the cure progressed, kept alive the myth for six years. At last, when Wilson died, and the firmness and density of the aneurysm was ascertained to be solely due to contraction of the sac and condensation of its walls, the sac itself containing only *post-mortem* clots, then it was felt that another explanation must be sought more

<sup>1</sup> The case of Allison, p. 437 of the 2nd edition of this work, was a good example of this.

<sup>2</sup> Henry Cowan, aged thirty-eight, admitted 15th February, discharged 6th June 1872.

consonant with the actual facts. This explanation was not difficult to find. From the first it had been noticed that under the influence of the iodide the pulsations within the sac and even throughout the arterial system were much diminished in force, and in the second of our cases (XLII., p. 450) this was so marked as to attract the attention of the patient himself. This had been rightly referred to a reduction of the intra-arterial blood-pressure, but had been regarded as nothing more than an important adjuvant to the blood coagulation, hitherto the only recognised method of cure.

An aneurysm—you will remember—is formed because the normal blood-pressure suffices to overcome the resistance of some weak part in the arterial wall, which it gradually dilates with each pulsation till it ruptures. Lowering of the blood-pressure might stop further dilatation, but could not promote a cure unless the sac were simultaneously filled with coagula, or its walls became thickened and contracted. Experience during life has taught us the fallacy of the idea that the sac gets filled with clots, while the results of *post-mortem* examinations have shown that under the influence of potassium iodide coagula are only occasional and concomitant, and that the essential relief is due to contraction of the sac and thickening of its wall. In one case formerly referred to (at p. 389) the iodide treatment had been followed by marked benefit; the superficial pulsation had almost completely disappeared, and there had been a simultaneous lessening of all the pressure symptoms. This patient died under the care of a colleague from a different disease. On examination an aneurysm the size of a small orange was found to spring from the ascending aorta, and from this a smaller sac, the size of a walnut, projected anteriorly and was adherent to the thoracic wall in the third interspace. Not a trace of clot was to be found within the sac. Sections were made through various parts of the wall of this aneurysm, and on examining them microscopically the *intima* was found to be everywhere diseased, granular degenerated, and irregular in its thickness,

as if being worn away. The muscular layer of the *media* was tolerably dense, and apparently hypertrophied, over the large sac close to the artery; over the small sac at its external part the *media* was still present, but granular and undergoing fatty degeneration. The elastic layer of the *media* did not seem to be atrophied or degenerated in any part examined. The *adventitia* was everywhere considerably hypertrophied, but specially so over the outer part of the smaller sac.<sup>1</sup> This hypertrophy of the muscular coat, where that still exists, and of the *adventitia*, with concomitant contraction, are found in all aneurysms that have been treated with potassium iodide with any measure of success. We may refer this, if we will, to some hitherto unsuspected action of the drug upon the muscular and fibrous tissues, but it seems more rational to look upon this hypertrophy of the muscular and fibrous tissues, under the conditions present, as but another manifestation of that well-known law (Paget) by which a hollow muscle hypertrophies when opposed to an obstacle with which it is able successfully to contend. The facts narrated have been so often and so invariably observed that it is unnecessary to seek further proof, but I may mention that a Russian experimentalist named Bogolepoff has pointed out that one of the chief actions of potassium iodide is to lower the blood tension uniformly throughout the body by dilating the arterioles, the heart's action being at the same time diminished in force. In dogs this lowering of the blood-pressure and lessening of the cardiac force is frequently accompanied by increased frequency of the cardiac action, unless the dose is considerable, when slowing of the heart's action occurs as a preliminary to cardiac paralysis. In frogs a large dose dilates the arterioles and at the same time slows the heart without any preliminary stage of rapid action.<sup>2</sup> So far as the action

<sup>1</sup> Case of Murray, p. 389; and *Edinburgh Medical Journal* (June 1876), p. 1142.

<sup>2</sup> "Zur Frage der physiologischen Wirkung des Iodkalium," *Moskauer pharmacologische Arbeiten*, S. 125; and *Virchow's Jahresbericht* (1876), erster Band, S. 402.



on man is concerned, experience in the treatment of aneurysm, as also in connection with the treatment of the senile heart,<sup>1</sup> shows that we may accept uniform dilatation of the arterioles as the cause of that lowering of the blood-pressure which forms so important an element in the treatment of both forms of disease. The action of moderate doses on the human heart is to diminish both the force and the frequency of the pulsations, and this occasionally in a very marked manner. When the drug is given in such large doses as twenty or thirty grains twice or thrice a day, there is, however, frequently a rise in the pulse-rate, which does not appear materially to interfere with the ultimate improvement of the case, though it probably—and I think certainly—prolongs the treatment. In some few cases the action of the iodide is more injurious, and the heart's action becomes unduly quickened up even to 170 beats per minute. This rapid pulsation has always a bad effect on the aneurysm; none of those in which it occurred did well, and as this rapid action of the heart seemed, in accordance with Marey's law, to depend upon too free an escape of the blood into the veins,<sup>2</sup> it became necessary to prevent this. In the absence of any known ratio between any certain dose of the drug and a definite rate of escape through the arterioles, it is necessary to proceed by way of experiment, and to repeat this experiment in every case when treatment is first commenced. In this way we are able to obtain better results in a much shorter time than was possible under the haphazard method at first pursued. Following this course we put every patient to bed for a few days without further treatment, the pulse-rate being taken night and morning. So soon as the average pulse-rate in recumbency is accurately ascertained, ten grains of potassium iodide are given every eight hours. If the pulse-rate remains unchanged, the dose of the iodide may be increased in a few days up to fifteen or

<sup>1</sup> *The Senile Heart*, p. 275.

<sup>2</sup> *Physiologie Médicale de la Circulation du Sang*, p. 206, par le Dr. E. J. Marey, Paris, 1863. "Le Cœur bat d'autant plus fréquemment qu'il éprouve moins de peine à se vider."

more grains every eight hours, raising it by one grain each dose until the pulse-rate begins to rise. It is only rarely that we can increase the dose to fifteen grains without raising the pulse-rate; indeed it is common enough not to be able to increase the dose beyond ten grains without this taking place. Should the pulse-rate rise, the iodide is stopped for one or two days and then we go back to the highest dose that did not raise the pulse and continue with that dose. When the iodide has been given in this way the success attained has been quite remarkable; the cessation of pain, the lessening of the pulsation, and the general improvement have been so marked and so rapid as to leave no doubt that this is the true method of administration. Under the old haphazard method of large doses improvement of any consequence was rare before the lapse of six months; with the carefully regulated dose of the present day we obtain similar results in three months and often in less. Naturally we are not more successful in curing our patients; they still require more or less surveillance all their lives, and the occasional use of the iodide. But we are now entitled to say that the relief obtained is no mere matter of chance, but is largely proportionate to the period at which treatment is commenced. Thus if we get the patient early enough, while some part of the *media* still remains, the relief may be unusually rapid and the cure almost complete; while if we have nothing but the *adventitia* to rely upon, longer treatment may be required, and the relief may be less complete, though even in cases evidently of this character the improvement is sometimes very remarkable.<sup>1</sup> In all my experience I have only twice seen complete intolerance of the iodide. In one case the very smallest dose gave rise to rapid pulse and to severe neuralgic pains in the

<sup>1</sup> This explanation of the action of potassium iodide in the treatment of aneurysm will be found enunciated in the *Edinburgh Medical Journal* for June 1876, at p. 1142, and in the *British Medical Journal* for 5th April 1879, at p. 511, forming part of the discussion as to the treatment of internal aneurysms at the Glasgow Pathological and Clinical Society on 5th March 1879. It will also be found in the second edition of this work.

abdomen, even when the patient was in complete ignorance of what he was taking. In the other case the use of iodide was so constantly followed by copious herpetic eruption—so-called hydroa—that it had to be given up. Iodide of sodium had a precisely similar effect. This was the more to be regretted as in both cases the action of the iodide on the disease was markedly ameliorative.

In regard to adjuvant treatment, whatever is capable of reducing the frequency of the heart's pulsations without impairing the strength of the patient or vitiating the quality of his blood cannot but be an important adjuvant in the treatment of aneurysm. It has always therefore seemed to me a matter of considerable importance to enforce the recumbent position, as a certain means of attaining this important end. In the earlier days of the iodide treatment, when the dose given was undoubtedly too large, very much of the success attained seemed due to the perfect rest always insisted upon. Nowadays, when we know more perfectly the mode of action of the drug, recumbency is not so absolute a necessity, though it is always an advantage and ought to be insisted upon for the first six weeks or two months at least.

Aware of the evils of starvation on the one hand and the risks of plethora on the other, my patients were formerly put upon a somewhat restricted mixed diet—fish, chicken, or rabbit being given for dinner, at first at all events, as being more easily digested than beef or mutton and less likely to give rise to so-called biliousness. They were also advised to be moderate in regard to quantity. At present, I still give—at first—light articles of food, as less likely to disturb the digestion of one put to bed without fever or any ailment likely to lessen his ordinary healthy appetite, but otherwise no restrictions are put upon the patients. Potassium iodide is believed by some to destroy the albuminates in the blood,<sup>1</sup> and, whatever be the cause, there is no doubt that its continuous administration tends to emaciation, and there is also

<sup>1</sup> *Vide* Kämmerer, Virchow's *Archiv*, Bd. lix.

no doubt that better results have been obtained under a full diet than under a restricted one. A similar remark may be made as to drink. All ordinary non-alcoholic drinks may be freely given, as the iodide produces so free a diuresis that there is no risk of any hyperæmia from that cause. Alcohol in any form quickens the heart's action and is always injurious, and its use should never be permitted except when absolutely required, and then only temporarily.

I need hardly say that notwithstanding the great and manifest relief accruing from this method of treatment, positive cures can only be exceptional and of the rarest occurrence, but in all but the most exceptional cases we have a right to expect freedom from suffering and prolongation of life. Aneurysms may prove fatal in so many various ways that a tendency to cure can only be accepted when we have unequivocal proof that the aneurysm is shrinking in all its dimensions. Even then incautious exertion may rupture the sac at some weak point, and death may thus suddenly assail one who thinks he is being cured. Compression of important organs by a solidified sac may induce dangerous and even fatal complications; and under the most favourable circumstances, the occurrence of a large aneurysm springing from a large and important artery proves the existence of such a diseased condition of the arterial coats as will render the patient's life ever after more or less a precarious one.

Nevertheless the results obtained from the potassium iodide treatment are sometimes most marvellous, and they can be and are obtained in cases in which no other treatment is available.

In 1873 I had under my care a gentleman with a large aneurysm of the aortic arch, the main symptoms being a remarkable *vox anserina*, and a constant headache with a flushed and turgid face, the result of compression of the large veins at the root of the neck. Under the use of the iodide, without any other treatment, all these symptoms were so



greatly relieved, and even his voice so much improved, that this patient ceased to consider himself an invalid. Over-exertion subsequently caused a relapse of all his symptoms; his head and arms were so turgid, his quasi-apoplectic symptoms so severe, and his aneurysm so large that a late distinguished physician (Dr. Warburton Begbie) gave him only a fortnight to live. Yet this gentleman lived long after the doctor himself, and the last time I saw him was to be reproached for my mistaken diagnosis, as some medical friends had told him he had no aneurysm. Wonderful as the improvement was, the aneurysm was still to be detected; he lived for a year or two longer and then died of cerebral embolism.

Again, for many years I had charge of a middle-aged lady, who, when I first saw her, about eighteen years ago, had a fluid pulsation between the left scapula and the spine, arising from an aneurysm of the upper part of the descending aorta. This aneurysm had not only eaten its way through the ribs posteriorly, but had caused erosion of the spinal column, with pressure on the nerve roots, indicated by constant numbness and tingling along the nerves on the left side, with recurrent nerve-storms of acute pain which darted up the neck and side of the head, and down to the tips of the fingers and the points of the toes, ending at last in a fit of violent sickness. Unfortunately every narcotic tried increased so much this deadly sickness that one after another had to be discarded. By and by the iodide told; the pressure lessened, the sac contracted, and the suffering gradually ceased. After ten years' confinement to bed from intense suffering this poor woman was so relieved as to be able to go about like other people free from pain. The aneurysm could still be detected, many of the objective symptoms remained in a modified form, but the pressure symptoms and the suffering were gone. This lady died about two years ago from an attack of acute bronchitis, her aneurysm remaining quiescent to the last. It is difficult to imagine a more interesting case, one in which

the ameliorative power of the drug could be more severely tested, or one in which this remarkable property could be more triumphantly displayed.<sup>1</sup>

<sup>1</sup> This case, with one or two more of considerable interest, has been recorded in the *British Medical Journal* for 6th June 1891, at p. 1220.

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